THE JOURNAL OF DENTAL RESEARCH

(Continuing the Journal of the Allied Dental Societies)

Number 1: February, 1934

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(Nos. 2–6: April–December, 1934)

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FUTURE OF THE JOURNAL OF DENTAL RESEARCH

QUOTATIONS FROM ANNOUNCEMENTS IN THIS VOLUME

February: page 1. 1. The Special Committee of the [International] Association [for Dental Research], to which the *Journal's* offer of transfer of ownership had been referred—Drs. L. M. Waugh, F. S. McKay and A. H. Merritt—[*J. Den. Res.*, 1933, *13*, p. iii] recommended to the Council [of the Association] that the proposed gift be accepted.

2. The Council has voted to recommend to the Association that the proffer be accepted.

3. The Council [in anticipation of such acceptance] has appointed the members of the above-named Committee, and also the President (E. H. Hatton), the President-elect (J. L. T. Appleton, Jr.), the Vice-president (T. B. Beust), and the Treasurer (Bissell B. Palmer), a Committee of Seven to recommend to the Association the number and personnel of the board of editors and also such other matters affecting management as they may wish to suggest.

April: page 68. (8) At the general meeting of the [International] Association [for Dental Research] (March, 1934) . . . the Association voted. . . to accept ownership of the *Journal.* All conditions of further procedure and management were referred to the Council for study, decision, and early enactment. Successive issues of this *Journal* will reflect the evolution of the Council's expressed desires.

June: page 238. The Council [of the International Association for Dental Research] voted. . . [June 1934 to accept the recommendation of the above-named Committee of Seven]. . . that the management of the *Journal* be entrusted (a) to a board of editors to consist of one representative of each section, to be elected by the respective sections, this board to select its own officers, committees, and other agents; (b) the five chief active officers of the Association to serve as the Acting Board of Editors pending organization of the permanent board. . . and so notified the [twenty-six] sections, which are now electing their representatives. Meanwhile the existing management of the *Journal*, for the convenience and protection of the Association, is serving with the Acting Board of Editors in a procedure that will transfer complete management to the Association as soon as transitional uncertainties can be removed and the Association is ready to assume all responsibilities.

June, August, October, December: page 4 of the cover of each issue—from a letter to the honorary editors by the retiring management. We have endeavored to make the *Journal of Dental Research*, since its establishment in 1919, an exemplar of the best and most meritorious in non-proprietary publication of dental research. The honorary editors have contributed the strength of their approval and support of that effort, and have responded to many calls for assistance. We cordially thank you for the very valuable aid you have given. Your interest and sympathy have fortified the *Journal's* determination to attain its avowed objectives. Your coöperation, accredited in each issue as a public endorsement of our purpose, has facilitated the *Journal's* growth and enhanced its usefulness. The *Journal*, on your recommendation [*J. Den. Res.*, 1933, *13*, 521; Dec.], is about to pass into the complete control of the International Association for Dental Research, which will receive it as a gift from the original management. The Council has voted to place the *Journal* under the direction of a board of editors representative of, and to be selected by, the sections of the Association. Each of you, as a member of the Association, will have opportunity to participate in the recurrent selection of these editors. It is deeply gratifying to us, as we formally sever our past relationship, that the *Journal*, as the official publication of the Association, will continue to have the abiding interest and individual support of its former honorary editors.—*William J. Gies, Executive Officer, Board of Editors.*

ADDENDUM. The second title page (page ii) presents the names of the elected sectional representatives in the Permanent Board of Editors. This Board has voted to entrust the active editorial management to an Editor, Associate Editor, and Assistant Editor, these three officers to constitute its Executive Committee. The Permanent Board is now determining by ballot its choices for these three positions. The Board has also voted to create a Finance Committee and has added to its membership, to serve in this relationship, the three trustees of the Research Association's Endowment Fund: Drs. Bissell B. Palmer (Treasurer of the Association), Arthur D. Black, and Russell W. Bunting. It is expected that all remaining executive decisions will be voted before the next general meeting of the International Association for Dental Research, in Chicago, March 16 and 17, 1935, and that the Association's complete control of the *Journal* will be inaugurated with the issue for April 1935.

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General meetings have been held on this schedule: (1) New York, Dec. 21, '22. (2) Chicago, Mar. 7, '24. (3) Chicago, Mar. 20, '25. (4) Chicago, Mar. 26, '26. (5) New York, Apr. 19, '27. (6) Washington, Mar. 25–28, '28. (7) Chicago, Mar. 23–24, '29. (8) Toronto, Mar. 24–26, '30. (9) Memphis, Mar. 21–22, '31. (10) Columbus, Mar. 19–20, '32. (11) Chicago, Mar. 18–19, '33; adjourned executive session, New York, May 25, '33. *Next general meeting* (12): Chicago, Mar. 17–18, '34, in coördination with the eleventh annual meeting of the American Association of Dental Schools, Mar. 19–21.

Objects (*Const., Art. II*): . . . "(a) To promote broadly the advancement of active research in all branches of dental science and in related phases of the sciences that contribute directly to the development of dentistry, and which add to the knowledge of the mouth and teeth and of their relations to the body as a whole; . . . (b) to encourage and facilitate coöperative effort and achievement by, and mutual helpfulness among, investigators in all nations in every division of stomatology; (c) to the end, particularly, that dentistry may render cumulatively more perfect service to humanity." (The Association has been using "dentistry" and "stomatology" as synonyms.)

Membership: total (Feb. 15), 302. *Eligibility* (*Const., Art. 14, Sec. 1*).—"Any person who has conducted, and published an account of, a meritorious original investigation in dental science, or in any of the sciences contributory to stomatology, shall be eligible to membership in this Association, providing such person conforms to the recognized standards of professional ethics." *Election.*—New members may be elected *only at general meetings of the Association.* Membership in a section is secondary to membership in the Association as a whole. Removal of members from one geographic center to another does not affect their membership in the Association.

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Endowment fund (as of May 25, 1933): $1,093.70. There have been no expenditures from this fund. *Trustees*—The Treasurer; Arthur D. Black, Northwestern Univ.; Russell W. Bunting, Univ. of Michigan.

Divisions may be organized by the members in any nation. There are now divisions in Austria, Canada, Czechoslovakia, England, and the United States—5. *Sections* may be organized by the members in geographic centers within the divisions. There are now sections (23) in Vienna ('29); Halifax ('28), Toronto ('21), Winnipeg ('30); Prague ('32); London ('31); Ann Arbor ('23), Baltimore ('33), Boston ('20), Chicago ('20), Cleveland ('30), Columbus ('32), Louisville ('32), Minnesota ('28), New Haven ('30), New York ('20), Philadelphia ('28), Pittsburgh ('28), Richmond ('33), Rochester ('33), St. Louis ('28), San Francisco ('24), Washington, D. C. ('31). The names of the secretaries are given on p. 1 of this cover.

Sectional meetings in 1933–34 (*schedule subject to revision*). *Ann Arbor*—May ?. *Baltimore*—Oct. 24, Nov. 15, Jan. 31. *Boston*—Nov. 6, Jan. 29. *Chicago*—Oct. 27, Jan. 26, May 18. *Cleveland*—Feb. 12. *Columbus*—Mar. 9. *Halifax*—Nov. 16, Jan. 26, Mar. 9. *London*—Oct. 18, Dec. 6, Jan. 24, Apr. 18. *Louisville*—Jan. 12. *Minnesota*—Nov. 20, Jan. 22, Feb. 26, Mar. 26, Apr. 23. *New York*—Dec. 7, May ?. *Philadelphia*—Oct. 13. *Pittsburgh*—Oct. 2, Nov. 6, Dec. 4, Jan. 8, Feb. 5, Mar. 5, Apr. 2, May 7. *Prague*—Jan. 12, May ?. *Richmond*—Nov. 18, May 26. *Rochester*—Oct. 13, 17; Nov. 10, 24; Dec. 8, 22; Jan. 5, 19; Feb. 2, 16; Mar. 2, 16; Apr. 13, 27; May 11, 25. *St. Louis*—Nov. 14, Jan. 9, Apr. 10. *San Francisco*—Nov. 16, Jan. 24, Feb. 28, Mar. 28, Apr. 25. *Toronto*—July 17, Sept. 12, Oct. 20, Jan. 18. *Washington*—May 3. *Winnipeg*—Feb. 7.

JOURNAL OF DENTAL RESEARCH

Further Announcements Relating to Prospective Transfer of Ownership

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In our issue for December, 1933 (p. 521), it was stated (a) that proposals relating to transfer of ownership of the *Journal of Dental Research* have been received from three dental societies or groups of societies; (b) that a majority of the whole board of editors recommended that ownership be transferred to the International Association for Dental Research; (c) that, in conformity with the action thus decisively voted, the Association had been assured of our readiness to transfer ownership of the *Journal* to the Association as of January 1, 1934, or as soon thereafter as the Association might wish; (d) that the related correspondence between the Association and the *Journal* had been referred by President Hatton to a special committee of the Association for a report to the Council, "which in turn will present its conclusions and recommendations at the next general meeting of the Association in Chicago, March 17–18, 1934." The following events bearing on this situation have occurred since the aforesaid statement was written last December:

1. The Special Committee of the Association, to which the *Journal's* offer of transfer of ownership had been referred—Drs. L. M. Waugh, F. S. McKay and A. H. Merritt—recommended to the Council that the proposed gift be accepted.

2. The Council has voted to recommend to the Association that the proffer be accepted.

3. The Council has appointed the members of the above-named committee, and also the President (E. H. Hatton), the President-elect (J. L. T. Appleton, Jr.), the Vice-president (T. B. Beust), and the Treasurer (Bissell B. Palmer), a committee of seven to recom-

mend to the Association the number and personnel of the board of editors and also such other matters affecting management as they may wish to suggest.

4. The Council has appointed the General Secretary (the writer) to ascertain whether various dental societies would cooperate with the Association in the support of the *Journal*. The cooperation assured to date (February 21) is indicated below:

(a) The First District Dental Society of the State of New York has appropriated $1,000 for this purpose in 1934.

(b) The New York Academy of Dentistry has appropriated $1,000 for this purpose in 1934.

(c) The American College of Dentists has appropriated $1,000 for this purpose in 1934.

(d) The Academy of Stomatology of Philadelphia has subscribed for copies for at least 25 of its members.

(e) The Allied Dental Council (New York) has subscribed for copies for at least 25 of its members.

(f) The American Academy of Dental Science has subscribed for at least 25 of its members.

(g) The American Stomatological Association has subscribed for at least 25 of its members.

(h) The Robert R. Andrews Society for the Promotion of Research (Tufts Dental School) has subscribed for copies for all of its members.

The foregoing facts and conditions will be presented at the meeting of the Research Association on March 17. The Association's action will be reported in the next issue.

EFFECTS OF SALIVA UPON GASTRIC DIGESTION

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During the last hundred years, the rôle of saliva in gastric digestion has received attention from many investigators. Some (Stricker, Friedenwald, 1) claimed that saliva regulated flow of gastric juice by direct action upon the gastric mucosa. Others (Biernacki, Schuld, 1) pointed out that these effects occurred only while food remained in the mouth, where the causative agent or agents must be sought. It might be due to CO_2-content of saliva (Fricker, 1), to substances liberated during mastication (Schuld, 1), or to an internal secretion of the salivary gland (2). All these early surmises are unfounded (3, 4, 5, 6). The oral phase of gastric flow is reflex in origin, mainly through stimulation of taste buds (5, 6), and is now called "appetite" secretion. Carlson (6) found that this flow averages about 3.5 cc. per min., but may run as high as 7.8 cc. per min. Therefore it is definitely known that neither saliva nor salivary glands regulate flow of gastric juice.

Does swallowed saliva influence flow of gastric juice, and modify activity of the latter? Investigations of these questions have been carried out upon Pavlov-pouch dogs and upon human gastric-fistula cases. Sokolow (7) investigated effects of test-meals of saliva, distilled water, bile, and pancreatic juice upon Pavlov-pouch dogs. His results show that saliva has a greater stimulating effect than distilled water upon rate of secretion, while concentration of pepsin was about the same for both. Carlson (6), using human subjects with gastric fistulas, reported that saliva had no effect upon the secretory mechanism. He pointed out, however, that saliva somewhat influenced the character of gastric juice, mainly by dilution, and slightly by neutralization, the latter power being approximately equivalent to an 0.8 percent Na_2CO_3 solution. Bendixen and Angelowa (8), while investi-

gating effects of nicotine upon gastric secretion, studied those of swallowed saliva. One group of human subjects ejected the saliva while another swallowed it. Allowing for the dilutional factor, calculated upon the basis of 3.5 cc. of gastric juice per min., 300 cc. of wheat test-meal, and 0.94 cc. of saliva per min., they discerned a stimulating effect upon pepsin and free HCl. This, they thought, was due to stimulation of the gastric mucous-membrane by an unknown agent. Allaria (9) reported that saliva interferes with the curdling action of rennin in children. Rossi (10) indicated that saliva improves peptic activity during early digestion. Babkin and Komarov (11) have shown that dog submaxillary-gland mucin does not influence peptic activity, while gastric mucin inhibits. This difference, they think, is due to release from gastric mucin of a hydrolytic product having definite inhibitory action.

It was the purpose of this investigation to study effects of swallowed saliva upon gastric secretion from the standpoint of specific chemical and physical properties. This called for study of acid-neutralizing properties, and effects upon peptic activity. We outlined the solution of these problems through the following determinations: (a) rate of flow of basal and stimulated saliva; (b) acid-neutralizing power of basal and stimulated saliva; (c) effects of saliva test-meal upon gastric secretion; (d) reflex effects upon gastric secretion of chewing sweetened and flavored indifferent substance; (e) effects of swallowed saliva upon gastric juice after a test-meal; (f) effects of saliva upon peptic activity of gastric juice; (g) effects of mucin upon peptic activity.

Methods of Analysis. We analyzed gastric samples for peptic activity, total chlorides, and free acid by the following methods:

Pepsin. The Gilman and Cowgill (12) modification of the Gates method was used. With a Mayer dremmeter, a constant standard in the glycerin silver-suspension was maintained. In this way, it was not necessary to run pepsin controls for each analysis. Gastric juice, diluted 50 percent with Sörensen's glycine NaCl-HCl solution to maintain constantly a pH of 2, was put in a glass cell and a small film placed over the solution, gelatin-side down. The whole cell was sealed and kept in a thermostat for 10 min. at 25°C. The cells were then removed, and films washed and dried, and then compared in a

colorimeter against standard silver suspension. From the reading on the colorimeter and the graph of standard pepsin solution, the percentage of pepsin was computed.

Chlorides (total) were determined by a modified Van Slyke method. Standard solution of $AgNO_3$ was prepared in concentrated HNO_3 solution. A measured quantity of gastric juice was placed in a measured quantity of $AgNO_3$ solution in a 250 cc. Erlenmeyer flask and digested at 100°C. until clear. The solution was then back-titrated with standard KCNS solution, with ferric alum as indicator.

Free acid was determined by titration with 0.1 *N* NaOH solution, with di-methyl-amino-azo-benzene as indicator. (Acid Cl was subtracted from total, to give neutral Cl.)

PROCEDURES (DETERMINATIONS) OUTLINED ABOVE. (*a*) *Rate of flow of basal and stimulated saliva.* The subjects provided *basal* flow during a 15-min. period, emptying the mouth only when it was apparently full, to avoid stimulation produced by oral dryness; *stimulated* saliva was obtained by chewing gum for 15 min. *Basal flow* averaged 0.5 cc. per min. in 252 determinations on 15 subjects. There were individual and group variations of considerable magnitude. Possibly if subjects could be induced to eject saliva less frequently, mouth dryness as a stimulant would be greatly diminished, and individual variations much reduced. *Stimulated flow* averaged about 2.5 cc. per min.; the minimum being 0.9 cc., the maximum 7.0 cc., per min. Here, the individual and group variations were of greater magnitude than for the basal flow.

(*b*) *Acid-neutralizing power of basal and stimulated saliva.* To 3 cc. of saliva was added 5 cc. of 0.05 *N* HCl. Using Topper's reagent as indicator, the solution was back-titrated with 0.05 *N* NaOH. Saliva was collected 3 to 4 hours after meals. *Basal* saliva averaged (232 cases), in acid-neutralizing power, the equivalent of 0.017 *N* NaOH. *Stimulated* saliva, with few exceptions, ran higher in neutralizing power than basal specimens, being equivalent to about 0.025 *N* (0.01 percent) NaOH (221 determinations). Calculated on this basis, about 140 cc. of stimulated saliva would combine with the free HCl in 100 cc. of gastric juice at the height of secretion (free acidity of 35, i.e., no. of cc. of 0.1 *N* NaOH required to neutralize 100 cc. of gastric juice, using Topper's reagent as indicator).

These results agree fairly well with other estimations already reported. Cohn (13) found that whole saliva had acid-neutralizing properties varying from 0.002 to 0.048 percent NaOH. Zebrowski (14) obtained much higher figures for parotid saliva—0.087 to 0.256 percent NaOH. Carlson noted that during mastication, average gastric flow was 3.5 cc. per min.; we have found it about 2.5 cc. per min. Early in the digestion cycle, saliva markedly dilutes gastric juice, as well as combines with almost all free HCl. Bergeim (15) reports that action of ptyalin usually continues for 15–30 min. after a meal is finished, and that this action terminates immediately upon the appearance of free HCl. He also pointed out that protein digestion in the stomach is delayed while any considerable amount of colloidal starch is present, because it adsorbs pepsin. Therefore, acid-neutralizing power of saliva may be said to have a two-fold benefit: combining with HCl to permit prolongation of ptyalin activity, whereby, all starches being changed either into maltose or soluble dextrins, adsorption of pepsin is kept at a minimum. Probably disturbance of the latter relationship induces symptoms (diarrhea, flatulence, etc.) so often associated with high-carbohydrate diet, for carbohydrate in feces in such cases may not be more abundant than in normal subjects.

(*c*) *Effects of saliva test-meal upon gastric secretion.* The subject swallowed an Einhorn tube and the stomach was emptied of its contents. Gum was chewed until 100 cc. of saliva were collected; meanwhile gastric removals were made at 15-min. intervals. The collected saliva was put down the stomach tube, and pumps made every 15 min. until the secretion picture returned to its basal state. Seven out of eight tests showed stimulating effects upon the acid-secreting glands. Free acid varied from a peak of 17.5 in one case to 50.0 (*table 1*) in others. This peak appeared in from 30 to 45 min.—as soon as diluting and neutralizing effects were ended (i.e., by ejection from the stomach and combination with free acid). The fact that the saliva test-meal contained both sugar and flavoring material from the chewed gum raises the question whether the saliva itself was responsible for the acid stimulation. In *table 1*, samples 0–0‴, it is evident that reflex stimulation of gastric secretion occurred while gum was being chewed. In the following procedure (*d*) this is verified.

(*d*) *Reflex effects of chewing sweetened and flavored indifferent sub-*

TABLE 1

Chlorides in gastric samples: neutral, free, and total

Subject: Gillis. Trial no. 17. Date: June 28, 1932. Test meal: 120 cc. saliva. Time started: 10:00 a.m. Remarks: Gum chewed, saliva ejected; samples 0′ to 0‴; saliva meal after 0‴.

SAMPLE NO.	TIME	SAMPLE	$AgNO_3$	KCNS	Cl IN 100 CC.	SAMPLE	FREE ACID IN 100 CC.	ACID Cl IN 100 CC.	NEUTR. Cl IN 100 CC.
	min.	*cc.*	*cc.*	*cc.*	*mg.*	*cc.*	*units*	*mg.*	*mg.*
0	0	2	7	5.15	277	2	0	0	277
0′	10	2	7	4.65	300	2	30	106	204
0″	20	2	7	3.50	387	2	22.5	80	307
0‴	35	2	7	4.05	350	2	12.5	44	306
1	50	2	7	7.20	139	2	0	0	139
2	65	2	7	5.20	274	2	35.0	124	150
3	80	2	7	3.75	368	2	50	177	191
4	95	2	7	3.80	370	2	7.5	26	344
5	110	2	7	4.65	310	2	0	0	310

TABLE 2

Pepsin content

Subject: Olsen. Trial no. 14. Date: July 11, 1932. Test meal: None. Time started: 12:30 p.m. Remarks: Basal and appetite juice.

SAMPLE NO.	BATH		READING		PEPSIN			JUICE (CC.)
	In	Out	Init.	Final	Act.	Aver.	Percent	
1	1:26½	1:36½	24.3	23.3	1			
1′	1:27	1:37	24.8	21.2	3.6	2.3	.088	Basal; 14 cc.
2	1:47	1:57	24.8	20.8	4.0			
2′	1:48	1:58	24.7	21.6	3.1	3.5	.11	Basal; 15 cc.
3	2:05	2:15	25.4	21.9	3.5			
3′	2:06	2:16	24.3	20.8	3.5	3.5	.11	Basal; 10 cc.
4	2:29½	2:39½	24.1	23.5	film spoiled			
4′ (gum)*	2:30	2:40	24.2	20.7	2.5	2.5	.09	Basal; 13 cc.
5	3:22	3:32	23.8	15.0	8.8			
5′	3:22½	3:32½	25.2	16.0	8.2	8.5	.32	Appetite; 30 cc.
6	3:28	3:38	24.0	15.2	8.8			
6′	3:28½	3:38½	24.5	15.0	9.5	9.1	.36	Appetite; 10 cc.

* Gum chewed during collection of 5, 5′, 6, and 6′.

stances. An Einhorn tube was passed and the stomach emptied of all contents in four subjects. Withdrawals were made every 15 min. for 30–45 min., to ascertain average basal secretion in a 15-min.

period. Each subject then chewed pieces of gum successively until all flavor and sweetness was lost. All saliva was ejected. Usually, three 15-min. "pumps" were made after gum chewing was started. *Table 2* gives typical results. Average basal secretion of gastric juice in a 15-min. period was 13.6 cc., about 0.9 cc. per min. Gum chewing increased this average to 32.0 cc. in 15 min., about 2.1 cc. per min. Carlson, in sham feedings, found the average appetite secretion to be about 3.5 cc. per min. Effects of gum chewing upon pepsin content of gastric juice are more striking than upon quantity of juice secreted.

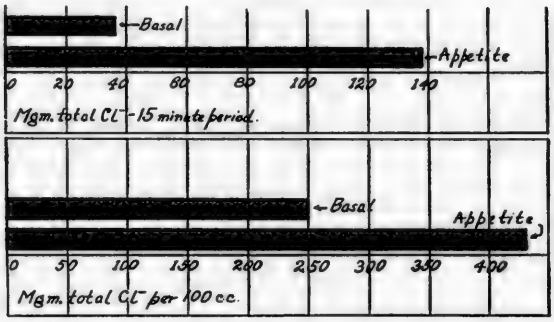
Basal
Appetite
0 20 40 60 80 100 120 140
Mgm. total Cl⁻ - 15 minute period.
Basal
Appetite
0 50 100 150 200 250 300 350 400
Mgm. total Cl⁻ per 100 cc.

FIG. 1. GRAPH SHOWING SECRETION OF TOTAL CHLORIDES

In a 15-min. basal period, the juice contained about 15 mgms. of 1–10,000 pepsin[1] as compared to 68 mgms. in 15-min. secretion following chewing of gum.

Carlson reported that 100 cc. of appetite juice, obtained from a case of gastric fistula, contained about 35 mgms. of 1–3,000 pepsin,[2] as determined by the Mett-tube method. Using the photographic-film method, 100 cc. of our appetite juice contained 340 mgms. of 1–10,000 pepsin. Recent investigations by Babkin and Komarov (11) may explain this difference. They found that among the end products of hydrolysis of gastric mucus were substances capable of markedly inhibiting peptic action. Such a substance is mucoitin-sulphuric acid.

[1] 1 gm. of 1–10,000 pepsin is capable of digesting 10,000 gms. of dried egg white in three hours.

[2] 1–3,000 pepsin: capable of digesting 3000 times its weight of dried egg white in three hours.

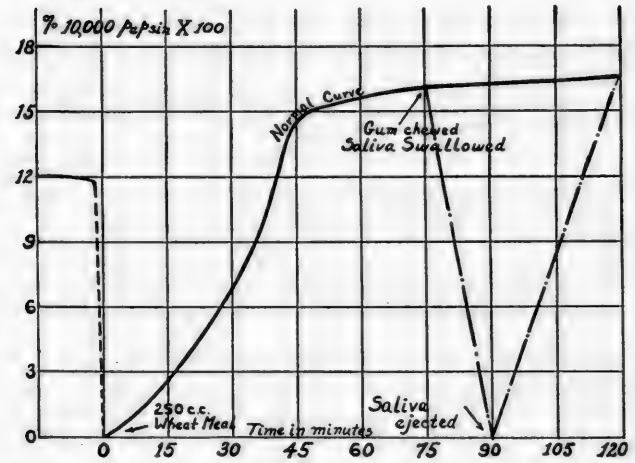

FIG. 2. GRAPH SHOWING EFFECTS OF SWALLOWED SALIVA UPON PEPTIC ACTIVITY AFTER A TEST-MEAL

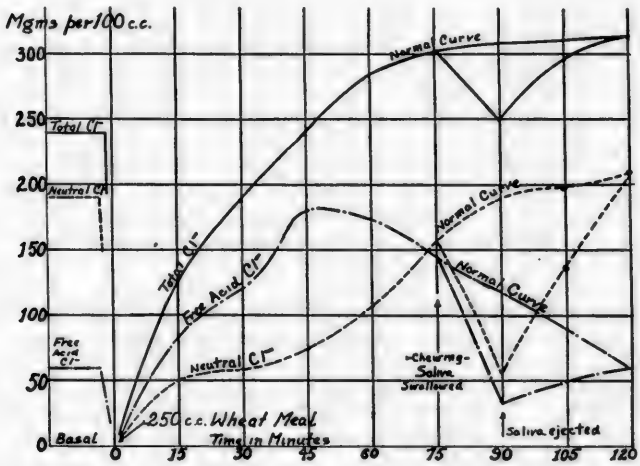

FIG. 3. GRAPH SHOWING EFFECTS OF DILUTION AND CHANGE OF pH, FROM SWALLOWED SALIVA, UPON GASTRIC SECRETION AND PEPTIC ACTIVITY AFTER A TEST-MEAL

The Mett-tube method is usually carried out for 24 hours, sufficient time for release of considerable inhibitory substance from mucus. As a result, the Mett-tube method would give much lower figures for

concentration of pepsin in whole gastric juice than gelatin-film methods, which require only 10 min. for their operation. Very singular was the fact that the quantity of pepsin in all our 15-min. extractions of appetite juice calculated to 68 mgms. of 1–10,000 pepsin. The chloride graph (*fig. 1*) indicates similar stimulating effects upon total Cl.

(*e*) *Effects of swallowed saliva upon gastric juice after a test-meal.* A test-meal of 250 cc. of cream of wheat, or 5 percent alcohol, was given. Withdrawals were made every 15 min. for about 2 hours as described in (*a*). About twenty controls were run; then the subjects

TABLE 3

Chlorides in gastric samples: neutral, free and total

Subject: Gillis. Trial no. 7. Date: Mar. 26, 1932. Test meal: 250 cc. cream of wheat. Time started: 10:00 a.m. Remarks: No breakfast; gum chewed and saliva swallowed after 1 and 3.

SAMPLE NO.	TIME	SAMPLE	$AgNO_3$	KCNS	Cl IN 100 CC.	SAMPLE	FREE ACID IN 100 CC.	ACID Cl IN 100 CC.	NEUTR. Cl IN 100 CC.	REMARKS
	min.	*cc.*	*cc.*	*cc.*	*mg.*	*cc.*	*units*	*mg.*	*mg.*	
0	0	2	8	7.65	199	2	0	0	199	
1	15	2	8	10.20	32	2	0	0	32	
2	30	2	8	8.9	115	2	0	0	115	Gum
3	45	2	8	7.6	248	2	32.5	115	133	
4	60	2	8	7.6	248	2	15	53.3	194.7	Gum
5	75	2	8	7.35	220	2	30	107	113	
6	90	2	8	8.40	149	2	7.5	26.6	122.4	
7	105	2	8	8.45	145	2	0	0	145	

were given flavored and sweetened chicle to chew, and the saliva swallowed. This was carried out at various intervals after the ingestion of the test-meal. As had been anticipated from *in vitro* experiments, acidity of gastric contents was modified both by dilution and neutralization. Peptic activity was markedly inhibited as a result of dilution and change in pH (*figs. 2 and 3*). Local effects of the saliva were sufficient to overcome reflex stimulating effects of the chewing shown in (*d*). *Table 3* shows effects of swallowed saliva during the first hour after a test-meal. At the peak of the acid curve, swallowed saliva reduced free, neutral, and total Cl, the effect being greatest upon the free-acid Cl as a result of both dilution and neutralization. *Fig. 3* shows that it took about 45 min. for the saliva to be removed

from the stomach, when the curves returned to normal. During the actively secreting gastric phase (first hour after the test-meal), the effects of swallowed saliva disappeared in 15–20 min. (*table 3*). In experiments where saliva was swallowed during the gastric after-

TABLE 4

Effects of saliva upon peptic activity of 0.5 percent (1–10,000) pepsin solution; pH controlled at 2.75

PEPSIN	SALIVA	COLORIMETRIC CHANGE IN FILM	AVERAGE CHANGE	PEPSIN CONTENT
per cent		*mm.*	*mm.*	*per cent*
0.5	Buffered, no saliva	12.1 12.5 12.5	12.3	0.5
0.5	25 percent saliva buffered	11.7 12.4 11.7	11.9	0.45

TABLE 5

Effects of 0.5 percent mucin solution upon peptic activity of 1–10,000 pepsin: Gates gelatin-film method

PEPSIN	MUCIN	COLORIMETRIC CHANGE IN FILM	PEPSIN CONTENT
per cent	*per cent*	*mm.*	*per cent*
1.0	0	15.3	1.0
1.0	0.5	14.2	0.8
0.5	0	11.4	0.5
0.5	0.5	10.3	0.38
0.25	0	9.7	0.25
0.25	0.5	9.0	0.20
0.12	0	4.7	0.12
0.12	0.5	4.5	0.11
0.06	0	3.6	0.06
0.06	0.5	1.1	0.03

secretory phase, return of free acid and total-Cl contents to the basal state was no slower than in cases where none was swallowed. This indicates that saliva was ineffective as a stimulant during that gastric phase.

(*f*) *Effects of saliva upon peptic activity of gastric juice.* Gastric juice, buffered to a pH of 2.75, was mixed with quarter, third, and

half proportions of saliva and ensuing peptic activity determined. *Table 4* shows typical results. There was slight inhibitory effect, activity dropping from that of 0.5 percent to 0.45 percent. We cannot state what factor in saliva is responsible for this inhibition, nor the nature of the process.

(*g*) *Effects of mucin upon peptic activity.* A 0.5 percent solution of gastric mucin (Fogelson) was made in Sörensen's glycine, NaCl-HCl buffer (pH-2). Activity of 1–10,000 pepsin in percentages of 1, 0.5, 0.25, 0.12, and 0.06 was determined by the modified Gates method. Fresh solutions were made up for each determination, so that little hydrolysis of mucin could occur before use. *Table 5* shows that gastric mucin moderately inhibits peptic activity. There was a reduction of about 20 percent. Results obtained by Babkin and Komarov show inhibition varying from 30 to 75 percent. Their data indicate that where free acidities were the same, inhibition ran between 30 and 40 percent. Where a 75 percent impairment was shown, free acidity (Töpfer's Reagent) of the control was 44, while that of the mucin sample was 16. We are checking various factors involved in the mucin effect, and will report results at a later date.

CONCLUSIONS. (1) Basal flow of saliva averages 0.5 cc. per min., possibly somewhat less. Stimulated saliva flows at the rate of about 2.5 cc. per min.

(2) Acid neutralizing power of basal saliva showed greater group than individual variations, and compared on the average with 0.017 *N* NaOH. Stimulated saliva usually ran higher than the basal, and approximated that of 0.025 *N* NaOH.

(3) Chewing of sweetened and flavored indifferent substances resulted in "appetite" secretion of about 2.1 cc. of gastric juice per min. This juice showed high content of pepsin—340 mgm. of 1–10,000 pepsin per 100 cc. as compared to 35 mgm. of 1–3,000 pepsin reported by Carlson. The Mett-tube method does not accurately measure concentration of pepsin in gastric juice because, during the necessary 24 hours for the test, there is progressive release of substances inhibitory to peptic activity. The Gates gelatin-film method offers a more accurate method of estimating pepsin concentration because the process requires only 10 min.

(4) Swallowed saliva affects concentration of the constituents of gastric juice by dilution, and free HCl by neutralization in addition.

Of gastric juice, 100 cc. would be neutralized by 140 cc. of stimulated saliva at the height of the secretory curve.

(5) Saliva test-meals show stimulating effects upon the acid-secreting glands. Presence of sugar and flavoring material in saliva was probably largely responsible for most of the stimulation.

(6) Saliva influences peptic activity directly as it changes the pH of gastric juice. Maintaining a constant pH in the solutions, saliva was found to exert slight inhibitory effect (10 percent).

(7) With constant pH, gastric mucin exerts upon peptic activity a somewhat greater inhibitory influence than saliva.

(8) Saliva, by combining with free HCl in the stomach, early in the digestion cycle, promotes continuance there of the action of ptyalin for some time after entrance of food into the stomach. Indirectly, this enhances the gastric digestion of proteins.

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(13) Quoted from the Babkin Monograph, p. 48 (4 above).

(14) Quoted from the Babkin Monograph, p. 48 (4 above).

(15) BERGEIM, O. Salivary digestion in the human stomach and intestines, *Arch. Int. Med.*, 37: 110, 1926.

INFLUENCE OF DEVITALIZED HETEROGENEOUS BONE-POWDER ON REGENERATION OF ALVEOLAR AND MAXILLARY BONE OF DOGS[1]

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Many investigators have observed the influence of local conditions on healing and regeneration of bone in cases of fracture and loss of that tissue. Such local factors as blood supply, physio-therapy, and bone grafts, have been used. Live autogenous bone grafts have been advocated as conducive to the most successful result because dead and heterogeneous bone is more liable to be cast off as a foreign body, and live and autogenous bone is more compatible with the host. Besides, in live grafts osteoblasts, which are necessary for regeneration, are supplied. Schroeder[2] reported the use of $(Mg \cdot CaH_2PO_4)_2$ as an aid in root implantation. Kauffer[3] mixed homogeneous boiled bone with lanolin, castor oil and petrolatum, to the consistency of bismuth paste, and injected it into fractures of maxillary bones. He reported nine successful cases. Six were injected on or before the fourth day of injury. Three, non-union cases of four-weeks' standing, have definite experimental value. Murray[4] found, in experiments on radii of dogs, that autogenous boiled bone produces union; he also had success with an artificial source of bone salts. Murray wrote, in part:

"If we resect 2.5 cm. of a dog's radius and throw it away, leaving the gap in bone to heal as it will, we get a fibrous union filling the gap between the two bone ends, with a little ossification just adjacent to the bone-ends where we have killed some little bone by our operative procedure. If,

[1] Preliminary report. Presented at a meeting of the Faculty, School of Dental and Oral Surgery, Columbia University, March 15, 1933; also, of the New York Section, International Association for Dental Research, May 25, 1933.

[2] Schroeder: *Correspondenzblatt für Zahnärzte*, 1911 xl, p. 271.

[3] Kauffer: *New Jersey Dental Journal*, 1915, iv, p. 429.

[4] Murray: *Minnesota Medicine*, 1930, xiii, p. 137.

instead of discarding the resected portion, we remove all periosteum and endosteum, and break it up into chips and place *them* in the gap, we get a solid bony union; although, as we have seen from our sections, these bone chips are dead and autolyzed, at least in part. If, to make sure of their death, we boil the fragments before putting them back, we get bony union notwithstanding. If we decalcify these fragments before we put them back, *we get no evidence of bony union.* Obviously, viability . . . . and everything else except the calcium being disposed of as a factor, the rôle of these bone chips seems to be to supply calcium for the calcification of the granulation tissue constituting the healing process. Therefore, I have resected in other dogs the same amount of radius, discarded the fragment, and placed in the 2.5-cm. gap powdered sterile $CaCO_3$ and $Ca_2(PO_4)_3$—supplying an artificial source of calcium in the gap previously filled by bone—and *solid bony union has resulted.* Similar procedure, using sterile, powdered, calcium hexose phosphate, has again given solid bone in the 2.5-cm. gap."

In attempting to relate Murray's work on long bone to regeneration of alveolar and maxillary bone when lost in advanced cases of alveoloclasia, cyst formation, cavities resulting from osteomyelitis, etc., it was foreseen that practical difficulties would hinder use of live autogenous bone—that in this effort it would be necessary to procure fresh bone from the host for each treatment of all affected teeth in cases of alveoloclasia, or of cysts on both sides or in opposite jaws. It was believed, therefore, that either an artificial source of bone salts or heterogeneous non-vital bone would meet the difficulties. The latter source was used in this series of experiments on alveolar and maxillary bone of dogs.

General procedure. All cases were done under general anesthesia. Incision was made, tissue retracted, and mallet and chisel used for removal, from each side of the jaw, of a box-like area of buccal plate of alveolar process about 1 cm. square, by 2 to 4 mm. deep. On the side used for control, the mucosa was merely sutured in place; on the other side, bone-powder was packed densely and thoroughly incorporated with blood, so that the subsequent clot would act as scaffolding and binder for the powder. The mobility of teeth was not disturbed at any time. Interrupted sutures were used in all cases. No therapeutic agents or drains were used. Eight animals were operated, two of which were discarded because of suppuration at the site of operation, on both control and experimental sides. The time from

date of operation to opening for clinical examination was from three to nine weeks. Histological examination followed.

Control procedure: upper left. Denuded roots of cuspids or premolars of bone and cementum, producing cavity 10 x 12 x 2 mm.; merely sutured lips of wound to place. After five weeks, area presented muco-periosteum healed and firmly attached to underlying tissue. Depression could be palpated in bone beneath mucosa. Incision and retraction revealed deposition of new bone at margins of box, but remaining area of box was filled in with fibrous tissue (*fig. 3*). (Control side of each of six animals presented essentially same clinical picture, at end of experimental period.)

Experimental procedure: upper right. Removed bone, 10 x 12 x 2 mm., denuding roots of cuspids or premolars. Inserted powder of long bone of sheep. After five weeks, area presented muco-periosteum healed and firmly attached to underlying tissue. No depression could be palpated. Incision and retraction revealed original box-like area filled in with hard firm substance, apparently early bone formation, which was continuous with surrounding bone, level with it, and indistinguishable from it (*fig. 1*). (Experimental side of each of six animals presented essentially same clinical picture, at end of experimental period.)

Histological data of typical case. The tissue blocks of control and experimental animals were decalcified, embedded in celloidin, sectioned, and stained with haematoxylin and eosin. A description of specimen 775, treated with bone powder (control, no bone powder), follows: Examination showed (*fig. 2*) that removal of the alveolar plate, cementum, and part of the dentin of the root resulted in new formations in all areas. The control (*fig. 4*), on which the same type of operation had been performed, showed no new bone formation. A thick *alveolar plate* was formed, almost filling in the entire area from which the cementum and dentin had been removed. It appears to be about double the thickness of the original plate. The newly formed bone takes haematoxylin stain somewhat more intensely than the rest of the alveolar process, reacting like young bone. Bone formation was still active at the close of the experiment, as indicated by the great number of osteoblasts covering the surface of the new alveolar plate. Bone formation was stimulated in adjacent, untouched areas of the alveolar process, as shown by thin layers of new bone covering the core of older tissue. The operated area of the *root*, in which an appreciable amount of dentin had been removed, showed at all places

FIG. 1

FIG. 2

FIG. 1. *a*, Radiopaque area on *experimental* side of jaw, due to early bone formation; after five weeks. Dog 775.

FIG. 2. *Experimental* side of jaw. *a*, Osteoid tissue replacing original cementum. *b*, New peridental membrane. *c*, Dentine. *d*, New alveolar bone. Dog 775.

FIG. 3

FIG. 4

FIG. 3. *a*, Radiolucent area on *control* side of jaw; after five weeks. Dog 775

FIG. 4. *Control* side of jaw. *a*, Dentine. *b*, Original cementum. *c*, Fibrous tissue. Dog 775.

layers of an osteoid tissue of variable thickness, making the surface of the root more smooth. The first reaction to the operation appears to have been resorption, Howship's lacunae later having been repaired by osteoid tissue. In some areas there is a suggestion of connective-tissue fibers incorporated with osteoid tissue, thus apparently re-attaching the root to the surrounding bone.

Summary. It seems obvious that placement of heterogeneous devitalized bone-powder into surgical cavities, in jaw bones of dogs, produced new bone formation. These cavities only simulated pathology in human jaws. No local complications developed excepting suppuration on both control and experimental sides of two dogs, the causes of which are unknown. The authors have made use of devitalized bone-powder in cases of cysts and alveoloclasia. The results, concordant with those in dogs, are not sufficiently numerous as yet to warrant definite conclusions.

We are indebted to Dr. H. J. Leonard, through whose efforts this research was made possible, for extremely valuable suggestions throughout the study; to Dr. C. F. Bödecker, for the histological report; to Dr. Houghton Holliday, for assistance in radiographing specimens; and to Miss Nesta Hughes, for the preparation of the histological specimens.

VELOCITY OF SOLUBILITY OF VARIOUS SAMPLES OF DENTAL ENAMEL

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Introduction. The resistance of teeth to acid decalcification has been investigated in an attempt to explain known variations in immunity and susceptibility of individuals to dental caries. Miller (12) observed that individual human teeth appeared to have different rates of decalcification in acid broths. Mummery (13) found, when freshly extracted teeth were placed in 0.075 percent lactic acid and the enamel examined daily with a probe for evidence of decalcification, that (a) enamel of various individuals differed in susceptibility to decalcification; (b) cusps and incisal edges decalcified first; and (c) hypoplastic enamel decalcified at a rate no greater than the average for all teeth studied. Using a gravimetric method, details of which are open to criticism, Pickerill (14) concluded that teeth varied in resistance to acids, and that enamel of unerupted teeth was nearly twice as rapidly soluble as that of erupted teeth. Pickerill's method required the tested teeth (paraffine coated) to be perfectly dry before and after immersion in acid, a precaution he did not seem to observe. McIntosh, James and Lazarus-Barlow (10), in experiments designed to determine whether teeth vary in power to resist acids, obtained inconclusive results. Enright, Friesell and Trescher (7) covered with asphaltum all the surface of whole teeth excepting small areas on the enamel, and exposed them to citrate and lactate buffers of different degrees of acidity for different periods. Upon histological examination, they found that enamel from the same individual and from different individuals varied in resistance to acids.

It is believed by many that "hardness" or "softness" of enamel may be an important factor in determining rate of solubility in acids, and therefore may be associated with dental caries. These terms (hard and soft teeth) are frequently encountered in dental literature, but

have not as yet been carefully defined, since the physical constants of enamel have not been completely described. Black (3) investigated, gravimetrically, the moisture, organic and salt contents of dentin, and found continuous increase in density of dentin from youth to old age. He concluded that neither density of teeth nor their percentage of salts has anything to do with susceptibility to caries. Karlström (8), in experiments on hardness and density of enamel by physical methods, found it hardest at incisal edges and cusps, and progressively less hard as the cervical margin is approached. Karlström reported that permanent teeth are about 7 percent harder than deciduous teeth. Richter (16) noted that deciduous enamel is softer than that of permanent teeth, and that enamel of pyorrheic teeth is harder than that of normal teeth. Dobbs (6), attempting to correlate enamel hardness with resistance to acid decalcification, concluded that these properties are associated. He observed that unerupted teeth lacked the resistance to acid decalcification common to erupted teeth, and concluded that enamel after eruption is changed in some unexplained manner, thus increasing its resistance to the action of acid. Dobbs found that immersion of teeth in a solution of trypsin at body temperature removed the acid-resisting properties of the surface of their enamel, but that these properties were not affected by brushing; results suggesting that resistance to decalcification may be associated with presence of organic plaque and (or) enamel cuticle. He found, further, that cusps showed changes first, and that enamel areas exhibiting resistance to weak acids were covered by plaques. His conclusion that resistance of enamel to acids is associated with hardness and density is not justified, since he seems to have proven that variations in rate of solution are associated not with hardness, which was not measured, but with presence of accumulated material on the surface. In addition, were hardness and rate of solution of enamel directly associated, it would be difficult to understand why cusps and incisal edges of teeth decalcified first (Dobbs and others), for these are the hardest portions of enamel, as reported by Karlström. Exact, comparable measurements of rate of decalcification of enamel have not been made by previous workers. The following experiments were planned to determine rate of solubility of various specimens.

Technic and methods. The technic employed is essentially that reported in a previous publication (1). A freshly extracted tooth of known history was cleansed of visible debris with scaler and water, and dried in an oven at 100°C. for approximately 72 hours. Enamel, then chipped from it, was carefully inspected for particles of dentin which, when found, were removed by grinding. The particles were powdered in a mortar, and the powder sifted through a 100-mesh phosphor-bronze screen sieve. Fifty mgms. of the powder, weighed to ±0.1 mg., were transferred to a test tube. To this were added 10 cc. of acetate buffer having a pH 4.60 checked by the quinhydrone electrode method, and prepared according to directions in the International Critical Tables (vol. 1, p. 81). The test tube, stoppered with paraffined cork, was kept in a shaking machine for from 5 minutes to 1 hour, when the tube was removed and contents promptly filtered. Two cc. portions of filtrate were analyzed in duplicate for Ca by the Clark-Collip (1925) modification of the Kramer-Tisdall method. Two cc. portions of the same filtrate were also analyzed in duplicate for P by the microgravimetric method of Pregl (15). Blank tests were made of the buffer for Ca and P. In general, enamel was reacted for 60 minutes. At longer periods each enamel would approach the same limit; i.e., saturation of buffer at that pH. Some 15-minute determinations were made in addition to 1-hour determinations, to obtain the slope of the curve of solubility.

Fifty mg. of enamel powder used in the foregoing procedure were dissolved in 0.1*N* HCl, and in a volumetric flask diluted to 50 cc. Ca and P were determined in duplicate, to ascertain whether variation in rate of solution of enamel is reflected in composition or in Ca:P ratio. These findings were also used to determine whether any change in proportions of Ca or P, or in Ca:P ratio, took place with changes in age.

By this procedure, rates of reaction of enamel are determined like those of any salt. While there is no available evidence to indicate that surface enamel differs in chemical composition from internal enamel, this method determines in each case rate of solution of enamel of average composition, and also reduces the influence of structural differences among enamels, or within any individual sample.

Data. Fig. 1 indicates results of shaking finely ground enamel in acetate buffers of pH 4.60 for periods of from 5 to 60 minutes. The following effects are evident: (a) During the first 5-minute exposure to the buffer, there was sharp rise in rate of solution. Different samples showed different rates of decalcification at the end of this interval.

(b) At the end of 15 minutes, rate of solubility became slower per unit time, and more nearly uniform for the different specimens. (c) This uniformity in rate of solution occurred between the 15-minute and 60-minute periods. It has previously been shown (1) that difference in size of the particles, and consequently in area exposed to the buffer, may result in as much as 20 percent difference in rate of solubility. This probably accounts for variations in rate at the early

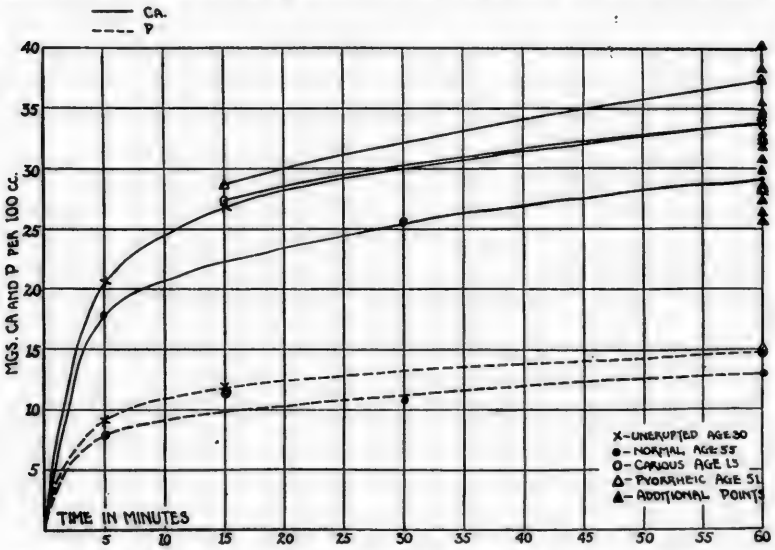

FIG. 1. GRAPH SHOWING VELOCITY OF SOLUBILITY OF VARIOUS SPECIMENS OF HUMAN ENAMEL

period. Having passed the early variable period (first 15 minutes), the powders dissolved at rates which were nearly uniform, as is indicated by parallelism of the curves. It is probable that the wide scattering of points recorded at the 60-minute period could be reduced by a refinement in technic that would make the area of the powders more nearly equal. This could be accomplished with two sieves of slightly different mesh, using only enamel that passed through one and not the other. However, with the small quantities of material available from each tooth this selection was impracticable.

In addition to determination of solubility rates, the effects of age, condition of tooth (i.e., carious, pyorrheic, normal, hypoplastic, or unerupted), and number of existing carious teeth at time of extraction, were studied in reference to total amounts of Ca and P in each sample of enamel. The data (*table 1*) show that, with the methods used and few teeth studied, (a) Ca and P contents did not increase with age; (b) within narrow limits, Ca and P contents were quite uniform; (c) deciduous enamel (five specimens varying from 6 to 11 years of age) did not contain less Ca or P than permanent enamel (fifteen specimens varying from 10 to 67 years of age); (d) the specimen from an unerupted tooth did not differ in Ca or P content from other specimens; (e) two hypoplastic specimens also contained average quantities of Ca and P. Since it was impossible to correlate incidence of caries with any other observed factor, it is not included here.

Discussion. These results differ from those of Bernardi (2), who found, in enamel of bulls 2–5 years and 7–10 years old, that Ca increased about 20 percent in this age interval whereas P remained constant. Toverud (18), finding that whole molars and depulped incisors of guinea pigs on deficient diet showed reduction in Ca and P, concluded that changes in structure and chemical composition of teeth may precede decay. Toverud did not determine incidence of caries in his animals. It is obvious that the osteodentin formed while an animal is on a deficient diet alters the total ash yield of a tooth, but not necessarily of the enamel, since the osteodentin is probably higher in organic content and lower in inorganic content than normal dentin. In addition, the abrasion that usually takes place on occlusal surfaces of molars in such experiments reduces the quantity of enamel and consequently the ash yield. Much has been written (4, 9, 11) concerning the relationship between susceptibility to dental caries and structural excellence of a tooth, and influence of diet on each. The evidence for this relationship, as presented, is in some measure dependent upon Ca and P contents. Conclusions in this field are influenced by a consideration that has been largely neglected: the fact that a tooth is not homogeneous tissue, and that analyses of teeth are greatly influenced by the proportions of enamel and dentin, limits the significance of all experiments (in relation to enamel caries) which

TABLE 1

Data relating to enamel from teeth of known age and condition

TOOTH[1]	AGE	STATE[1]	TIME: MIN.	ANALYSIS OF BUFFER (100 CC.)					ANALYSIS OF ENAMEL (50 MG.)				
				Ca		P		Ca:P	Ca		P		Ca:P
				Mgm.	Avge.	Mgm.	Avge.		Mgm.	Avge.	Mgm.	Avge.	
5d	6	C	60	25.56 25.56	25.56	11.18 11.47	11.33	2.26	20.09 20.27	20.18	8.71 8.79	8.75	2.30
4d	7	C	61	26.35 ...	26.35	11.33 11.40	11.36	2.32					
5d	9	C	60	27.50 27.65	27.58	11.39 11.43	11.41	2.41					
5d	9	C-h	60	32.58 32.74	32.66	13.80 14.09	13.95	2.34					
6	10	C	15	23.89 24.04	23.97	10.02 9.88	9.95	2.42					
			60	30.61 31.16	30.89	12.94 13.16	13.04	2.36	20.81 20.84	20.83	8.71 8.78	8.74	2.38
5d	11	C	60	29.78 ...	29.78	11.47 12.49	11.98	2.48	20.88 20.98	20.93	8.88 8.88	8.88	2.36
6	11	C	60	28.08 28.71	28.40	12.13 12.35	12.24	2.32	20.12 20.39	20.25	8.64 8.75	8.70	2.31
6	12	C	60	31.38 32.14	31.76	12.94 13.14	13.04	2.43	20.05 20.21	20.13	8.61 8.86	8.73	2.31
6	13	C	15	27.04 ...	27.04	11.11 11.11	11.11	2.43	21.30 21.29	21.30	8.78 8.80	8.79	2.42
			60	32.44 33.04	32.74	13.73 13.87	13.80	2.37					
6	13	C	15	27.21 27.26	27.24	11.25 ...	11.25	2.42	20.79 20.94	20.87	8.64 8.70	8.67	2.40
			60	33.43 33.59	33.51	14.45 15.25	14.85	2.26					
6	13	C-h	60	34.25 34.58	34.42	14.60 14.67	14.64	2.35					
6	15	C	15	32.44 32.50	32.47	13.36 13.43	13.40	2.42	20.94 20.97	20.96	8.75 8.77	8.76	2.39
			60	40.12 40.75	40.43	17.65 ...	17.65	2.29					
6	17	C-h	60	32.26 32.29	32.28	13.43 13.50	13.47	2.40	21.05 21.07	21.06	8.64 8.66	8.65	2.43
8	30	U	5	20.61 20.75	20.68	9.22 9.36	9.29	2.23					
			15	27.04 ...	27.04	12.05 ...	12.05	2.25	19.48 19.62	19.55	8.30 8.43	8.37	2.34
			60	34.00 34.14	34.07	14.45 15.40	14.93	2.28					
7[2]	33	P	60	28.15 28.20	28.18	11.04 11.11	11.08	2.53	21.23 21.34	21.29	8.75 8.76	8.76	2.43
8[2]	33	P	60	28.59 28.61	28.60	12.27 12.35	12.31	2.32	20.95 20.97	20.97	8.72 8.79	8.76	2.40
4	51	P	15	28.75 28.81	28.78	11.26 11.91	11.59	2.48					
			60	37.53 37.56	37.55	15.03 15.18	15.11	2.48	20.61 20.67	20.64	7.99 8.93	8.46	2.44

TABLE 1—*Concluded*

TOOTH[1]	AGE	STATE[1]	TIME: MIN.	ANALYSIS OF BUFFER (100 CC.)					ANALYSIS OF ENAMEL (50 MG.)				
				Ca		P		Ca:P	Ca		P		Ca:P
				Mgm.	Avge.	Mgm.	Avge.		Mgm.	Avge.	Mgm.	Avge.	
8	55	N	5	17.92 18.11	17.97	8.06 8.13	8.10	2.22					
			15	22.27 22.30	22.29	9.80 9.95	9.88	2.26	19.52	19.58	8.82	8.82	2.22
			30	25.52 25.64	25.58	10.53 10.60	10.57	2.38	19.64		8.82		
			60	28.94 29.17	29.06	13.14 13.14	13.14	2.21					
5[2]	67	P	60	38.41 38.30	38.36	16.12 16.27	16.20	2.36	20.99 21.00	21.00	8.89 ...	8.89	2.36
6[2]	67	P	60	34.68 34.75	34.72	14.09 14.31	14.20	2.42	20.06 20.26	20.16	8.24 8.24	8.24	2.44

[1] Meaning of symbols: *C*, carious. *d*, deciduous. *h*, hypoplastic. *N*, normal. *P*, pyorrheic. *U*, unerupted.
[2] From the same mouth.

seem to show that whole teeth are influenced by dietary or other systemic or local changes. There is little evidence to indicate that chemical composition of enamel is related to dental caries.

The increasing density of enamel with age, as reported by Karlström, may not be due to changes in Ca or P content, nor to variation in Ca:P ratio. This effect may be one of dehydration, with consequent increase in density—a condition that would not be detected by the methods used in the present study. That hardness and rate of decalcification are not related is indicated by the foregoing results, since enamel from permanent teeth, which are harder than deciduous teeth (Karlström), decalcified at the average rate of deciduous enamel.

Ca:P ratios of whole enamel average 2.38. This is somewhat higher than that calculated for enamel as the complex salt described by Gassman, and Taylor and Sheard (17), and others—$(CaCO_3)n \cdot Ca_3(PO_4)_2$—where n is between 2 and 3. However, in dealing with the small quantities used for the analyses described above, the accuracy of the available methods is not sufficiently great to justify any conclusion relating to probable composition of enamel.

Summary and conclusions. Comparison was made of velocity of solution, and total Ca and P contents of powdered, dried, enamel from teeth of individuals of various ages—6 to 67 years. The results indi-

cate that, within narrow limits, the Ca and P contents of human enamel are quite uniform, and the rate of solution and the Ca:P ratio bear no apparent relationship to age or state of the teeth.

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DISINFECTION OF PULP CANALS AND PERIAPICAL TISSUE WITH SURGICAL DIATHERMY[1]

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Electrotherapy has gained a place of prominence in the field of medicine, and recently has attained increasing value in dental fields. Diathermy, a form of electrotherapy, generates heat within the tissues, removed from the place or location of its applicators. It is a current of extremely high frequency, the modern machine yielding a higher amperage at a lower voltage (high-frequency currents of *quantity*), the older types yielding a lower amperage at a higher voltage (high-frequency currents of *tension*). Generally speaking, with the help of diathermy, we are able to administer heat wherever it is desirable—to any area, superficial or at any required depth. Diathermy is used in two forms: (a) Medical or constructive diathermy, which applies to any heating *within* physiological limits; and (b) surgical or destructive diathermy, which applies to any heating *beyond* physiological limits, producing a destruction of tissue. In the work covered by this report, heat capable of producing destruction of tissue was employed. The applicators were a sheet of lead foil, 14 in. square, placed on the chair as the inactive electrode; and a smooth broach as the active electrode. A milliampere-meter, graduated from 0–150, was used as a gauge. The *technique* employed for the treatment of cases presenting a necrotic and putrescent pulp-canal follows:

(a) Isolate the tooth with a rubber dam, the operating area of which has been painted with tincture of iodine. (b) Have all instruments and dressings sterilized. (c) Gain convenient access to the canal; open into the pulp chamber and remove its contents. (d) Saturate a paper-point with contents of the canal, and transfer same to ascitic agar medium, semi-solid, for incubation. In cases where the canal is dry, a paper-point is passed

[1] Preliminary report. Proceedings of the International Association for Dental Research: *Journal of Dental Research*, 1933, xiii, p. 206.

to the apex, moistened with the exudate, and transplanted to the medium. (e) Insert the active electrode slightly past the orifice of the canal, and apply current for five seconds by count after the tolerance point of the patient has been reached.[2] (f) Advance the applicator about one fourth the length of the canal, and repeat the treatment. This is continued until the apex has been reached and the heat is generated within the tissues beyond. After an interval of a minute or two, the treatment is repeated, starting from the apex toward the pulp chamber. Of all cases treated up to this time, none received more than three applications at one sitting. Since a hot arc is produced when the walls of the canal are dry, the canal is flooded with either normal salt solution or chlorine solution. This conducts the heat through the canal, and eliminates the crackling noise so evident to the patient, who tends to give an erroneous tolerance point for the heat. (g) The canal is now thoroughly dried, especially where chlorine has been used as a conduction medium; the canal curretted with broaches and files; and a culture placed in incubation. (h) A paper-point with a trace of eugenol is now placed in the canal, and the tooth sealed with crown and bridge cement. The next appointment with the patient is made after forty-eight hours, when the results of the cultures have become known. If the cultures previously made are positive, the treatment is repeated. Where the cultures are negative, the tooth is opened, the dressing removed, and a culture from the apex incubated. After the canal has remained negative for a week without further treatment, it is filled with gutta-percha cones, sealed, and x-rayed. The root fillings are placed to conform with the technique advocated by Dr. J. R. Blayney and taught in this College. Follow-up x-ray pictures are taken six months after the filling is placed.

Upwards of twenty-five cases have been treated with diathermy and, after a negative culture has been obtained, filled with gutta-percha cones. Thus far, the work has been restricted to root canals giving a positive culture before treatment; and included some that were unresponsive to routine treatment, in the Infirmary, with curettage and drugs. The average number of treatments, consisting of from two to three applications per treatment in each case, totaled three. Where

[2] The milliampere-meter is graduated from 0–150. In most instances the patient begins to feel heat at 40–60 m. a., but the tolerance point is not reached until about 70 m. a. The amount of milliamperage necessary, however, varies with resistance of the patient, and amount and rapidity of conduction of heat (from the area of treatment) by the blood stream, lymph, and other body fluids, and by the tissues themselves. In many instances as much as 110 m. a., were employed before the patient's tolerance point was reached.

the size of the foramen permitted, cultures were taken from the area beyond the root apex. A majority of treated cases had only one canal. Those with multiple canals did not respond as readily to treatments, requiring an average of one or two more treatments. In most of the canals, it is possible to coagulate and remove necrotic débris, which otherwise might remain intact owing to insufficient curettage. In cases presenting a discharging sinus, it was noted that the sinus discontinued its discharge and healed over usually after the second treatment. In all cases, the sinus had healed before the canal was filled.

In cases where a gas-producing organism subjects a tooth to severe soreness, producing inflammation of the p.d.m., application of diathermy causes cessation of gas formation and permits sealing of eugenol as a medicament. The amount of current used in these instances was less than where no acute inflammation was present. In cases where cultures remained positive after application of diathermy, and where the long-chain gram-positive streptococcus was prominent before the application, this organism seems to have been broken up into many gram-positive diplococci. A further study of this finding should prove interesting. Recently, in opening into the pulp chamber of an upper central incisor with a dead pulp and an infection cyst, there was a flow of exudate containing both long- and short-chain gram-positive streptococci, and gram-positive staphylococci. After the first treatment by diathermy, the bacteriological report was negative. At the second treatment, the culture taken *before* application of diathermy was positive, showing apparent decrease in the number of long-chain gram-positive streptococci and increase in the number of short-chain gram-positive streptococci, which in most instances arranged themselves as gram-positive diplococci. The culture taken *after* the second application of diathermy was negative, and remained so until the time for filling the canal. After the third treatment, the flow of exudate desisted and a slight hemorrhage was evident on the tip of paper-point placed in the canal to be transplanted in culture media. After a week of negative culture, the canal was filled and the root resected.

Not enough time has elapsed to permit study of more than one case six months after treatment. This was an upper left cuspid, with a

history of dead pulp under a large synthetic filling, presenting an acute alveolar abscess. An incision was made for drainage, the pulp chamber opened, and the patient dismissed. Within two days the swelling had subsided and the tooth was only slightly sore to percussion. A pledget of cotton with a trace of cresol was sealed under cement. The following day, the patient having returned with a swelling, the filling was removed to permit drainage. The x-ray showed an upper left cuspid with a very large area of destruction (approx. 3 mm. × 5 mm.); also an upper left first bicuspid, with an incomplete filling, having an area of destruction at its apex. The latter was extracted, expecting removal of a seat of infection with subsequent satisfactory treatment of the cuspid. Within a week after this extraction, it was impossible to retain a medicament under sealing. After the first treatment by diathermy, a sealing was placed. Six months after filling the canal, the x-ray showed an area of destruction of only about 1.5 mm. × 1.0 mm.

This study has been clinical and limited. Some material now being prepared for microscopic study will enable us, we hope, to determine the extent of changes, either pathologic or physiologic in the dentine and in the soft tissue, subsequent to treatment with diathermy.

COMPOSITION OF CRYSTALLINE AMALGAMS OF ZINC, LEAD, TIN, AND CADMIUM

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Daniell (1) observed that a zinc (Zn) rod partially immersed in mercury (Hg) for ten days became pointed at the lower end and covered, on the immersed portion, with six-sided plates of amalgam. A year later he reported (2) that tin (Sn) and lead (Pb) reacted similarly, Sn forming "six-sided tables" and Pb "soft, feathery crystals." Horsford (5), repeating the experiment with Sn, reported a compound: $HgSn_8$. These crystalline amalgams were thought to be chemical compounds by early workers, who assigned many and varied formulas. The direct method of study was criticised by Ogg (12), who pointed out the impossibility of freeing the crystals completely from mother liquor by filtration which, with the high specific gravity of Hg, would occasion a large probable error in the results of analysis. Workers then turned to indirect methods. Reference is made below to a few of many investigations.

Kerp and Böttger (10) applied the phase rule to amalgams of Zn and cadmium (Cd). Their results in the case of Zn admitted of no definite explanation. As regards Cd, for the temperature range 0°–44°C. they found a solid phase in equilibrium with the liquid phase having all characteristics of a chemical compound corresponding closely to Hg_7Cd_2. Puschin (13) made thermal analyses and a metallographic study of amalgams of Zn, Pb, Sn, and Cd. He found no evidence for chemical compounds. Fay and North (3), from thermal analysis, worked out a diagram for the system Pb-Hg and inferred the compound $HgPb_2$. The diagram was further developed by Jänecke (6), who combined his own results with those of Fay and North, and of Puschin, and presented a diagram showing $HgPb_2$ and an eutectic quite close to Hg, f.p. about −40°C. Van Heteren (15) presented a diagram for the system Sn-Hg. This has been modified in view of

results obtained at the National Physical Laboratory. No compounds are indicated. Rosenhain (14) considers the diagram as still quite incomplete, and reports further work in progress. Hildebrand (4), studying liquid amalgams, made determinations of vapor pressure. He found no evidence for compounds of Zn, Pb, and Sn with Hg, but showed that his results could be explained by assuming the existence of HgCd in the amalgam together with free Cd. Von Simson (16) examined amalgams of Sn, Zn, Cd, and Pb by the x-ray powder-method. In amalgams of Sn, Zn, and Cd he found hexagonal lattices differing from those of the pure metals. Crystals of Pb amalgam (20%Hg) have the same face-centered lattice as pure Pb, contracted in dimensions by 1.6 percent. Mehl (11) studied alloys of the Cd-Hg system by the x-ray powder-method, but found no evidence for compounds.

The purpose of the present study was to ascertain how composition of certain crystalline amalgams is affected by varying some conditions under which they are formed. Necessarily the direct method was followed. Because of danger of disturbing by pressure whatever combination existed, the amalgams were not pulverized before analysis but only coarsely broken up. For these reasons the results can be accepted as approximations only, and close correspondence is not to be expected even in the case of parallel preparations. The metals were of high purity as shown by analyses furnished by the dealers, and were used as received. Recovered mercury was purified by repeated distillation by the Hulett method.

PROCEDURE. Rods of Zn, Pb, Sn, and Cd, and strips of silver (Ag) and copper (Cu), were partially immersed in Hg in 8-in. test-tubes. *Three sets* were prepared, with duplicate preparations (*a* and *b*) in each set. One set (C) was placed in a "cold" room, where the temperature was held at about 3°C.; a second (R) was held at room temperature; the third (H), in a "hot" room, temperature about 37°C. At the expiration of the "time" stated below, each set was removed, the crystals centrifuged over fine muslin as long as more than a trace of mother liquor was separated, and analyzed. A *fourth set* (O) was prepared by holding the metal in Hg at about 90°C. for 24 hours; a *fifth* (M) was obtained by heating Hg with about 5 percent of its weight of associated metal until the whole was liquid, and then cooling slowly.

An *amalgam of Sn* (E) was formed by making Hg the negative pole in a solution of stannous chloride as described by Joule (8). The crystals obtained in sets O, M, and E were fine and probably retained an exceptional proportion of mercury.

RESULTS. *Zinc amalgam.* Time: 99 days. Action especially rapid. Yield not measured, but greatest obtained. Closely packed mass of laminae. Some hexagons distinguished. Samples boiled 2–3 hours in HCl (1:1), Hg separated, and Zn determined with standard potassium ferrocyanide solution:

		C	*R*	*H*	*O*	*M*
Zn (percent)	*a*.....	39.83	43.64	44.28	47.21	39.28
	b.....	41.03	43.24	45.12		

Lead amalgam. Time: 160 days. Fine dendritic needles branching at right angles. Pb determined as sulphate:

		C	*R*	*H*	*O*	*M*
Pb (percent)	*a*....	64.08	65.63	62.26	61.29	60.00
	b....	64.16	62.69	64.14		
Yield (grams)	*a*....	2.7	7.4	7.0		
	b....	2.4	7.4	6.4		

Tin amalgam. Time: 193 days. Very regular hexagonal plates. Sn determined as oxide:

		C	*R*	*H*	*M*	*E*
Sn (percent)	*a*....	77.83	78.87	79.04	76.77	76.04
	b....	77.52	79.24	78.69		
Yield (grams)	*a*....	0.4	4.6	4.3		
	b....	0.4	4.7	4.3		

Cadmium amalgam. Time: 179 days. Stubby, rounded needles. Samples treated with hot HCl in contact with platinum, Hg separated, and Cd determined by volumetric method of Keffer (9):

		C	*R*	*H*	*O*	*M*
Cd (percent)	*a*....	14.50	15.67	16.54	21.94	15.63
	b....	14.84	15.09	16.57		
Yield (grams)	*a*....	8.0	23.2	28.8		
	b....	10.1	28.8	26.9		

Silver and copper. Beyond superficial amalgamation, almost no apparent action with Ag and Cu. Some minute points appeared on surface of Cu. From edge of Ag strip, few silvery threads projected. Analysis not attempted.

DISCUSSION. The appearance of the tube contents, at the close of the reaction, was in general as described by Daniell and Horsford (1, 2, 5). Immersed ends of rods had become pointed and, especially near the surface of the Hg, covered with crystals some of which were detached and floating in the liquid. It is clear that the metal dissolves in Hg; and that the resulting solution being lighter rises to the surface until saturation is reached, when crystals form about the rod. As the Hg most free from amalgam would be in the lower part of the tube, solvent action is there most rapid, and diminishes upward to the surface. The conical form assumed by the immersed part of the rods is thus explained. The characteristic crystals formed by each metal, differing little in composition though formed under varied conditions, suggest chemical action. Furthermore, the crystal-forming metals are all distinctly electropositive to mercury; and the speeds of reaction as measured by yield are, from greatest to least, Zn, Cd, Pb, Sn, which is their order in the electromotive series, except that Pb and Sn are reversed. In this connection it is recalled that aluminum (standing above Zn in the series), when adhering oxide is removed, amalgamates with marked vigor. The percentage values, when calculated to show atomic relations, correspond approximately to formulas assigned by others using the direct method. This is to be expected, and is of little significance.

Zinc. For crystalline Zn amalgam, Joule (8) found $HgZn_2$ (39.46%Zn). Results here reported correspond more closely to Hg_2Zn_5 (44.90%Zn).

Lead. The values for Pb, if a rather liberal allowance is made for retained mother liquor, seem to confirm the findings of Fay and North (3) and of Jänecke (6), who inferred from thermal analysis the compound $HgPb_2$ (67.38%Pb). Tentative work in this laboratory suggests an eutectic between Pb and this compound with a f.p. of about 160°C.

Tin. The percentage of Sn (about 79) is so high that no formula can be assigned with any assurance. It is not far from $HgSn_7$

(80.55%Sn), found by Joule (7). Horsford's value (5) was $HgSn_8$ (82.55%Sn).

Cadmium. The results for Cd accord in general with those of Kerp and Böttger (10), and appear to confirm their inference of a chemical compound in equilibrium with the liquid in the temperature range 0°–44°C. Their formula for this compound is Hg_7Cd_2 (13.76%Cd). My results, somewhat higher, correspond better to Hg_3Cd (15.73%Cd). Above 44°C., Kerp and Böttger found no solid phases showing compositions in accord with simple ratios. At 89°C., the solid phase contained 22.83%Cd. This is in fair agreement with my value of 21.94%Cd for crystals formed at about 90°C.

SUMMARY. (1) Zinc, lead, tin, and cadmium, immersed for many days in mercury, form crystalline amalgams containing the associated metal approximately as follows: Zn, 41–45%; Pb, 63–65%; Sn, 78–79%; Cd, 15–16%. Silver and copper under like conditions form almost no crystals. (2) Crystalline amalgams of these metals, prepared by several other methods, show compositions approaching the same values. (3) These results appear to confirm the findings of others as to the existence of the compounds $HgPb_2$, and Hg_7Cd_2 or Hg_3Cd. No such claim can be made as regards zinc and tin.

The author is indebted to Drs. V. C. Myers, J. W. Mull, and E. W. Skinner, for the use of laboratory conveniences.

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METHOD OF RECORDING FUNCTIONAL MOVEMENTS OF FULL-DENTURE BASES IN THREE DIMENSIONS[1]

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Since 1805, when Gariot built the first plain-line articulator, much work has been done on the problem of reproducing jaw movements on a machine. Many methods of recording mandibular movements have been devised; and many instruments have been invented in attempts to produce articulators capable of accurately reproducing those movements. These articulators are of two types: non-adjustable and adjustable. Non-adjustable articulators require only a centric-relation registration, and a proper adjustment of casts to the mechanism. The possible movements on this type are either arbitrary (Evans, Bonwill, Grittman, and Gysi Simplex) or theoretical (Monson, and Hall automatic anatomic). Adjustable articulators require multifarious registrations, the number and kind varying with the instrument. Articulators of this type are divisible into two groups: (a) two-dimensional instruments (Gysi adaptable, Hanau, Snow, and Wadsworth) and (b) three-dimensional instruments (Lentz, Hall, Stansbery, Phillips, Homer and Roberts). Registrations for adjustable articulators are taken either intra-orally or extra-orally. The principal intra-oral checkbite methods are the (a) interposed wax record of forward protrusion (Christenson), and of lateral occlusion (Waugh); (b) interposed metal bite-gages used with forward protrusion (Snow); (c) plaster checkbite (Stansbery); (d) dentographic registration (Luce); (e) carborundum biteplate method (Patterson); and (f) Gysi gothic arch tracings cut in compound bite-rims by pins in the opposing rim (Needles). The principal extra-oral methods are

[1] Presented at a joint meeting of the Cleveland and Columbus Sections of the International Association for Dental Research, Cleveland, Dec. 5, 1932. Also on the program of the general meeting of the Association, Chicago, March 18–19, 1933: *Journal of Dental Research*, 1933, xiii, p. 201.

the graphic (Walker), with the "facial clinometer" (further developed by Gysi and Phillips); and the extra-oral plaster checkbite (Sears).

All registration methods just named fail to give a complete functional record of the movements of the mandible, and of the relations of denture bases when in function. For our purposes, dentures are considered to be in function when there is actual tooth-contact in any relationship within the limits of tooth anatomy. By registering only positional or static relations, only a small part of the total functional relationship is recorded. By recording relations in only two dimensions, no provision is made for cusp height. Unequal compression of tissues is recorded excepting by methods that use a central bearing-point of contact between the ridges. A checkbite method, to be complete in registration of all relations of denture bases when in function, must record the correct centric relation—must register accurately all paths of the denture bases during the various excursions of the mandible—while the teeth are in contact. Any change in relationship of the bases must be recorded regardless of cause, if balanced occlusion is to be perfected on an articulator. It is my intention, in this presentation, to demonstrate an apparatus that makes these registrations, together with an articulator that reproduces the registrations, and thereby produces the correct relations of the denture bases in function. The *recording apparatus* consists of two triangular metal plates to be attached to the upper and lower biteplates. The upper metal plate has three cups attached at the corners, a central bearing-post, and an extension forward for a tracing pin. The lower metal plate has three small rounded tracing-posts in positions corresponding to those of the cups on the upper plate; a flat plug screwed into the center of the plate opposite the position of the central bearing-post of the upper plate; a tooth-form die, interchangeable with the flat plug; and a tracing tray located to correspond with the position of the upper tracing-pin (*fig. 1*).

Since denture bases do not travel in straight lines in function, any articulator that cannot reproduce the curved paths registered on the recording apparatus is inadequate. The *articulator* we are using consists of a tripod, the legs of which fit into three cups in a base. The upper cast is attached to a hinge suspended from the tripod; the lower, directly to the base. The legs of the tripod terminate in

articulating pins the same in size and shape as the tracing posts in the lower plate of the recording apparatus. An inner steel-cup is fitted and keyed inside each of the three holders on the base, each of which also has a keyed lid with a hole in the top near the rim. Into each cup is fitted a centering device for use in mounting the case in centric relation (*figs. 2* and *3*).

The *technic for the use of the recording apparatus* follows: Accurate impressions of upper and lower arches having been taken, casts are formed of material that does not disintegrate in vulcanization. Hard base-plates are formed to fit these casts, and the vertical opening for the individual case determined according to esthetic requirements. It is often advisable to set the twelve anterior teeth at this time; when this is done, a second set of bite rims are constructed for the purpose. By means of interposed wax, the casts are mounted on the articulator in centric relation. The wax is removed, and the hinge closed until the bite rims have approximately the same relation as in the mouth. The hinge is locked at this time, to preserve the correct vertical opening. The bite rims are cut down to make room for the recording apparatus, which is attached to upper and lower bite-rims in such a manner as to maintain the same vertical opening. The metal plates must be parallel—the upper directly centered over the lower. The flat plug is in place, and the tracing tray is prepared by smoking it over burning cajeput-oil and flowing carding-wax over this (*fig. 4*). The bite plates are then returned to the mouth, where the patient makes the various mandibular excursions necessary to trace the Gothic arch. Then by means of a plaster syringe, greatly accelerated plaster of Paris is forced between the two metal plates while the patient holds the tracing needle at the apex of the Gothic arch. Kerr's Snow White № 1 Plaster is satisfactory for this purpose, when mixed to a thin creamy consistency with a 50 percent solution of potassium sulphate (*fig. 5*).

When the plaster has set, the bite plates and plaster centric-checkbite are removed from the mouth, and seated on the lower cast on the articulator. The upper cast is removed from the upper member of the articulator, seated in the upper bite-plate, and reattached to the articulator. The casts are then considered as being in centric relation. The vertical opening is not changed, because the articulator hinge is locked and centering devices are in place. The plaster checkbite is then removed, and the flat plug in the lower metal-plate replaced by the tooth-form die, which corresponds to the mould of the posterior tooth to be used on the case. If the central bearing-post of the upper plate does not coincide exactly with the center of the tooth-

form die, the upper metal-plate should be removed from the bite rim and reset, so that the central-bearing post is in the center of the die and the

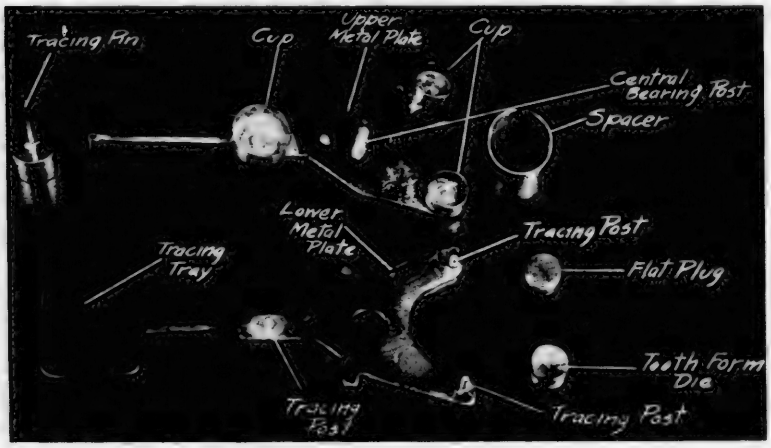

FIG. 1. RECORDING APPARATUS

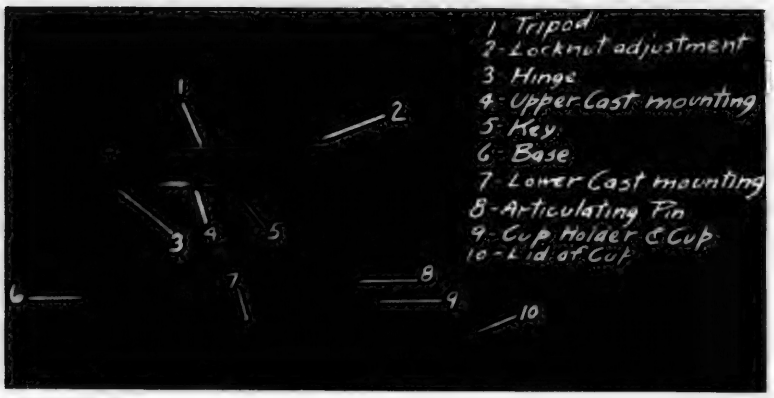

FIG. 2. ARTICULATOR

metal plates are exactly parallel. A spacer is used to locate this plate correctly. The three cups are then filled with modeling compound, and the three tracing-posts as well as the tooth-form die lubricated with cocoa

butter. The compound is chilled in place, and reheated with an alcohol blowpipe to a near-molten stage. The bite rims are carried at once to the

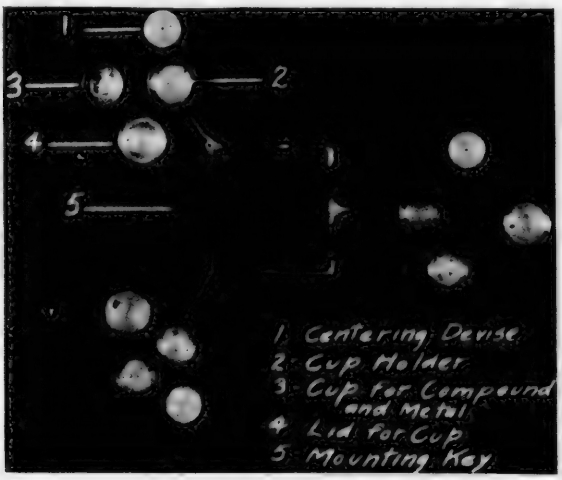

FIG. 3. BASE AND PARTS

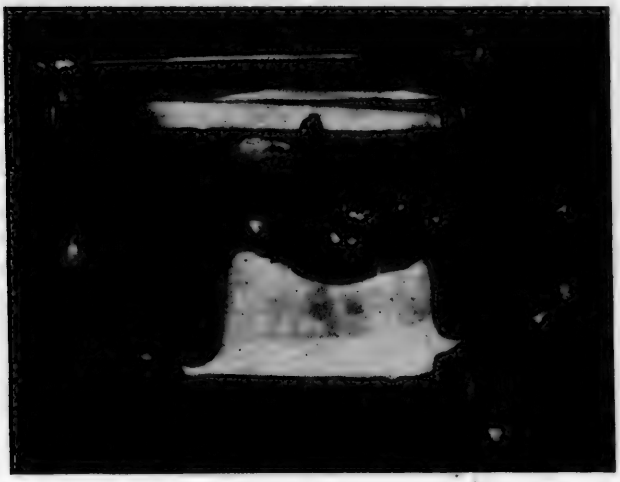

FIG. 4. RECORDING APPARATUS, SET UP ON ARTICULATOR, SHOWING SPACER IN PLACE

mouth, where the patient makes all possible motions of the lower jaw while keeping the central bearing-post in contact with the tooth-form die. This

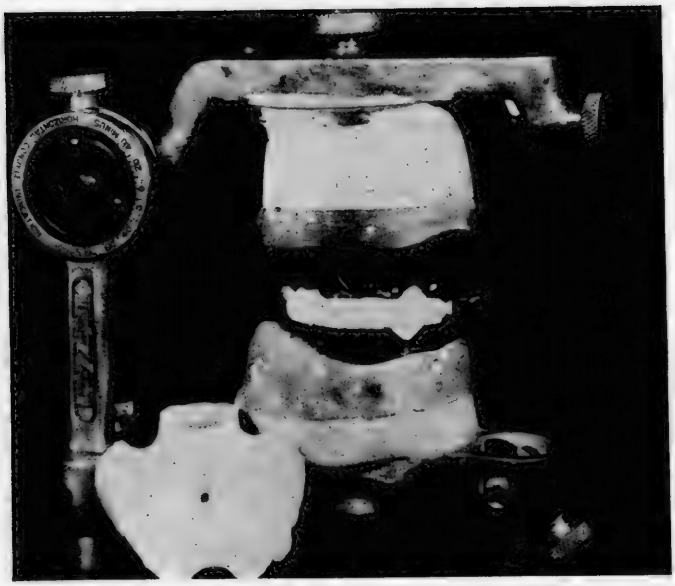

FIG. 5. PLASTER CHECKBITE, TO RECORD CENTRIC RELATION. (ARTICULATOR USED TO REPRESENT MOUTH)

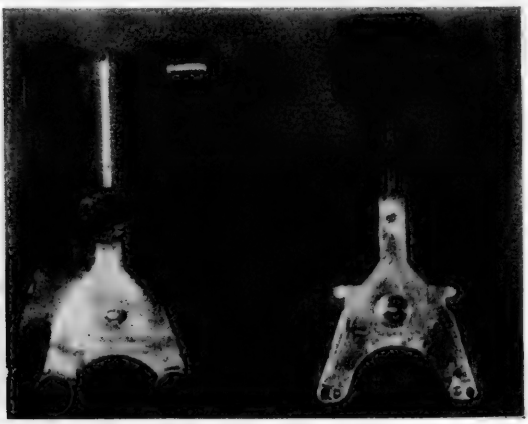

FIG. 6. TRACINGS IN CUPS MADE BY TRACING POSTS DURING VARIOUS EXCURSIONS OF MANDIBLE

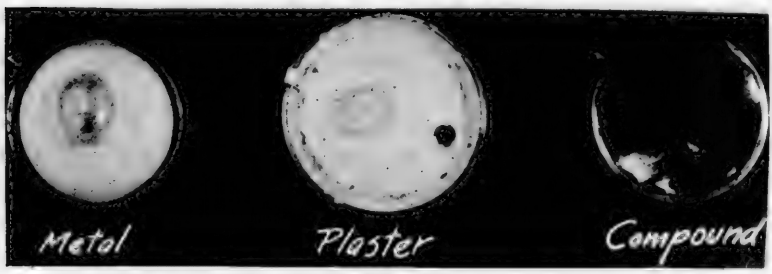

FIG. 7. TRACING IN MODELING COMPOUND; PLASTER IMPRESSION OF TRACING IN LID; AND TRACING REPRODUCED IN METAL

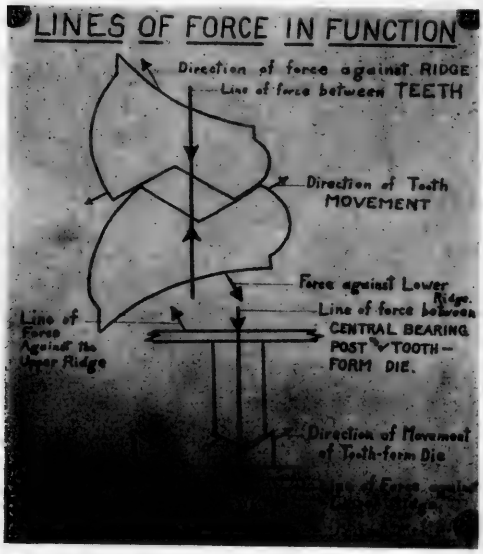
LINES OF FORCE IN FUNCTION
Direction of force against RIDGE
Line of force between TEETH
Direction of Tooth MOVEMENT
Force against Lower Ridge.
Line of force between CENTRAL BEARING POST & TOOTH-FORM DIE.
Line of Force Against the Upper Ridge
Direction of Movement of Tooth-form Die

FIG. 8. FORCE AGAINST RIDGES IS NOT VERTICAL

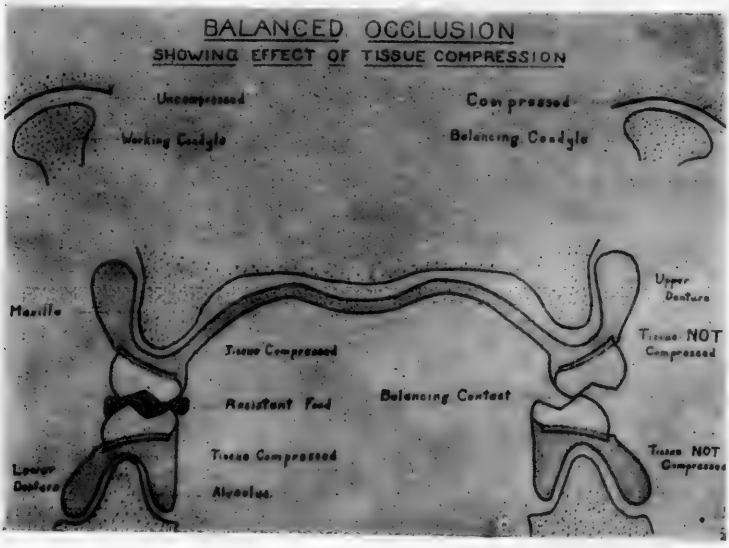

FIG. 9. BALANCED OCCLUSION IN FUNCTION

movement is in reality exactly the movement of the teeth in function. The records are made in the compound by the tracing posts (*fig. 6*).

A tracing may also be made on the tracing tray at this time, which is valuable only as a means to visualize what is going on inside the mouth. The patient continues moving the jaws until all protrusions are included; and then the compound in the cups is chilled in the mouth, *while the various excursions are still being made*, to reduce to a minimum distortion by shrinkage. The bite plates are then returned to the articulator, which is adjusted by means of the guides in the compound cups of the recording apparatus. The three centering devices are removed from the cups on the base of the articulator; the cups are filled with modeling compound and placed in position in the cup holders in the base; the articulating pins of the tripod are lubricated with cocoa butter. The compound is softened with the mouth blow-pipe almost to the flowing stage, when the tripod is brought back to position as guided by the tracings in the compound of the recording apparatus. It is then carefully moved about into all relations permitted by these tracings, corresponding tracings being made in the soft compound in the articulator cups. When this is accomplished, the compound in the cups is chilled, while the tripod is still in motion, and checked for accuracy. Then excess compound is trimmed off, and an impression of the compound tracing taken in the "lid" with plaster of Paris. Excess plaster, forced out through the hole in the lid, is removed when it is set, and a hole drilled through the plaster from the hole in the lid. The compound is removed from the steel cups, which are then replaced in the holders. Hooper's metal is poured into them and, before it can congeal, the lids are quickly thrust into position. Excess of metal escapes through the hole in the lid. Thus the tracings are reproduced in metal (*fig. 7*). The articulating pins of the tripod in contact with these tracings form the only movable joints of the articulator. They guide the casts in relation to each other through all possible relations of function. The teeth are now set up in balanced occlusion, and in correct relation to the ridges. The cases are waxed and vulcanized, and the casts preserved so that they may be returned to the articulator for correction of any errors developed during the process. The dentures are then finished and polished.

The technic just described is distinctive in several ways. (a) It records *paths* and not simply positions of the denture bases. These paths are in *three* dimensions as they *actually* are in function. (b) The registrations are entirely relative as to the bases, showing only the end result, or the combined effects of all known and unknown forces

involved in jaw movements and occlusion of dentures. The condyle paths, whether curved or straight, Bennett movement, compression of tissue, and shifting of the denture bases on the ridges, are disregarded as such; but the effects of these, and any other forces, are registered by the recording apparatus, and reproduced on the articulator. (c) The face bow is not essential in this technic, because the vertical opening is adjusted in the mouth, and not on the articulator. (d) Any shifting of the bases on the ridges is registered exactly as in function, since the same causative forces are involved, i.e., cusp height and inclined planes of the cusps. Positional registrations, even in three dimensions, are made under a direct vertical stress only. When teeth are in place, a lateral force as well is directed against the bases through the vertical force against the inclined planes of the teeth. *Fig. 8* shows the lines of force involved when the teeth are in function, and when the recording apparatus is making its registrations. (e) Balanced occlusion may be so accurately adjusted that a balancing contact is maintained when a bolus of food is interposed on the working side, within the limits of compression of the tissues on the ridges, and in the condyle fossae (*fig.* 9).

DENS IN DENTE

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"Dens in dente" (one tooth in another, or tooth inclusion) is one of the rarest malformations of human teeth. Since 1874, when this condition was first reported by Baume, not many more than a dozen cases have been described in dental literature, and several of these are recorded in such a cursory manner that exact conclusions as to structure and significance are impossible. Therefore, when I was fortunate enough to come into possession of a perfect specimen of "dens in dente," and to obtain serial histologic sections, it was necessary to study and analyze all previous cases of this kind. All reports bearing upon this problem will be enumerated in approximate chronologic order, and then discussed individually before describing my own case.

CASES OF DENS IN DENTE, OR RELATED DENTAL MALFORMATIONS, IN DENTAL LITERATURE[1]

Baume, 1874 (1877). Salter, 1875. Busch, 1897. Miller, 1901. Zeckendorf, 1910. Moral, 1918. MacDonald, 1918. De Jonge Cohen, 1918. Kirk, 1918. Lejeune and Wustrow, 1920. Bodenheimer,[2] 1921. Gnamm, 1922.[2] De Jonge Cohen, 1925. Erausquin et al., 1932. De Jonge Cohen, 1932. Hattyasy, 1933. Kronfeld, 1934.

Baume, in his Textbook of Dentistry (1877), illustrated a case of what he called "development of a tooth within another." This tooth (which had been described by Baume in the *Deutsche Vierteljahrschrift für Zahnheilkunde*, in 1874), an upper cuspid of unusually large dimensions, was removed from a patient whose upper lateral incisors had been congenitally missing. Therefore, Baume concluded that the abnormal dental tissues, enamel and

[1] See the alphabetic bibliography at the end of this paper for the original references, here and throughout the descriptions.

[2] I could not obtain, in Chicago, the original reports by Bodenheimer and Gnamm. Therefore, I am unable to consider Bodenheimer's case, which is only mentioned by Peckert (21); Gnamm's case, however, is described in detail by Peckert, and therefore, I shall use this reference for my discussion.

dentin, attached to the lingual surface of this cuspid represented the crown of the missing lateral incisor. This conclusion hardly seems justified in view of the fact that, in the abnormal dental structure contained within the cuspid, the enamel (d) lines a central cavity (f), whereas the dentin is found on the *outside* of this layer of enamel. If Baume's case were actually a union of lateral incisor and cuspid, we would expect, using our knowledge of united or fused teeth, to find the dentin *inside* of the enamel surrounding a more or less developed common pulp-chamber. Furthermore, Baume reports that the dentinal tubules of the cuspid surround the enamel crown of the included incisor tooth in a wide circle. This observation is almost conclusive evidence that the "included lateral incisor" of Baume is in reality an invagination of the enamel of the cuspid into the crown, very similar to the malformation of a cuspid described by Miller. Zeckendorf, doubting Baume's conclusions as to the fusion of lateral incisor and cuspid, suggested that absence of the lateral incisor was purely incidental, particularly since the cases of Busch, and Zeckendorf's own, showed that such a malformation may occur in mouths in which all other teeth are normally formed and in place.

Salter (1875) uses the term "warty tooth" for the malformation he studied, because this tooth, an upper lateral incisor, showed on its labial surface an irregular, warty lobulated mass. Salter prepared a labiolingual ground section, which revealed the presence of several small tooth-like structures projecting from the distorted outer wall of the incisor into the pulp chamber. The largest of these projections (plainly visible in his illustration) contains a heart-shaped cavity, which at first sight might be mistaken for a pulp chamber. However, Salter's description of the structure of this projection and a higher magnification of its wall disclose that the *inside* of this dentinal projection was lined with *enamel*. He stated that "on the outer surface (of the whole tooth, Ref.) is a layer of enamel, not only covering the exterior surface but also that (produced by the involution) of the heart-shaped cavity within it." Evidently Salter had the correct idea of considering these small, tooth-like structures not as fused additional tooth elements, but as a result of "the involution of the surface, which originally had within it many papillary and folded processes." Salter's case may be classified either as a dens in dente, if one considers the one tooth-shaped projection into the large common pulp-chamber; or, with Salter, as a form of odontoma, considering the presence of irregular masses of dentin, enamel, and vascular canals in other portions of the specimen. At any rate, presence of enamel on the inside of the dentin of the "inner" tooth is especially typical of all folding processes of the tooth surface (*figs. 1* and *6*).

The largest number of examples of dens in dente was reported by Busch in 1897. He was also the first to use the term, "dens in dente." Busch observed several typical cases,[3] considering them to be *twins* formed by one tooth's growing completely around another smaller one. He called special attention to the fact that, in the outer tooth, the enamel is found on the *outside;* in the inner tooth, on the *inside* of the dentin. He compared the relation of the two parts to two fingers of a glove, one of which has been turned inside out and then stuck into the other. Two cross-sections in different levels through one of Busch's cases are reproduced here; they show plainly the mutual relationship of the two tooth elements, and the arrangement of the enamel on the surface and on the inside (*fig. 1*, A, B). Busch's cases differ from those of Baume and Salter in that the formation of the inner tooth was almost identical with that of a true and independent tooth within the pulp chamber of the outer tooth. Busch considered dens in dente a special form of true twin-formation, whereby one twin completely surrounds the other. We shall see how this opinion—erroneous, by the way—can easily be conceived from exclusive study of *cross* sections through specimens in which nothing can be seen of the existing connection between enamel and dentin of outer and inner teeth.

One of the most interesting dental anomalies, which seems to give the clue to the dens-in-dente problem, was reported by Miller in 1901. He described a very large upper cuspid, the crown of which was divided into eight lobes by a series of grooves and indentations. Microscopic examination of successive cross-sections through the crown revealed that some fifteen columns of enamel were suspended, like stalactites, from the dome of the enamel caps, some extending into the root a quarter of an inch beyond the neck of the tooth. In cross sections these enamel columns appear roundish, triangular, or compressed, and are usually hollow, though some of them contain a core of very irregular osseous substance or a structureless mass, apparently resulting from deposit of calcified material in the remains of the enamel organ. The presence of some fifteen such enamel columns caused many individual centers of dentin formation; between some of these, dentin fissure-shaped pulp-cavities were found. In addition to this abnormality of the crown, a small root-like structure was found within and attached to one side of the wide-open root-canal. This root showed the remarkable peculiarity that cementum lined the *inside* instead of the outside. This

[3] It is difficult to determine the exact number of cases of dens in dente observed by Busch, because from his description it is not always possible to differentiate between dens in dente and fusions (twin formations). But at least three of his numerous cases seem to be typical.

inverted root represented continuation of one enamel column, and thus, like the crown, was turned inside out. Miller's case is interesting in many respects. Such an abnormality can be explained in no way other than as a folding of the developing tooth. This process of intussusception of one portion of the tooth into the other, with subsequent hard-tissue formation in the newly created abnormal position, can evidently occur more than once in the same tooth. In Miller's case, the enamel organ of the crown must have dipped into the pulp tissue at about fifteen different places. Nor is this invagination confined to the enamel-covered portion of the tooth, for the root of this same tooth contains another smaller root, which is also turned inside out.

Zeckendorf reported a case of dens in dente in 1910. The tooth, an upper lateral incisor of an adult, discolored and with pulp decomposed, had been extracted. Within the wide apical foramen another root-end with a fine apical opening was found. The tooth, sawed in two in a mesiodistal plane, shows, according to Zeckendorf, two root canals—one for the outer tooth, one for the inner (*fig. 2*). Zeckendorf suggested the term "dents télescopées" (telescoped teeth) in preference to Busch's term, dens in dente. Unfortunately, Zeckendorf does not report histologic findings in this tooth, nor does he give an exact account of the distribution of enamel in the outer and inner teeth. I especially regret this because Zeckendorf's case appears to be very similar to my own. If in his case the enamel were present on the *inside* of the inner tooth, I would be convinced that his case did not represent *two* "dents télescopées" but only *one* "dent télescopée"; namely, an invagination of crown into root, or a gigantically enlarged lingual pit.

The next report of a dens in dente is that published by Moral in 1918. His case was a supernumerary tooth in the upper jaw. In its pulp chamber there was a second tooth-like structure looking more like a true, small, peg-shaped supernumerary tooth than any other "inner" tooth published so far (*fig. 3*). Moral described and analyzed this case carefully, both macroscopically and microscopically. From the fact that the inner tooth contained enamel on its inside, and from the way it was attached to the outer tooth, he concluded that "*the anomaly known as 'dens in dente' is not a fusion of two teeth, but it is only one tooth. An extension projecting within the tooth toward the apex resembles an inner tooth.*" Moral recognized very clearly that the space within the inner tooth was not an inner pulp-chamber, but a space formerly occupied by enamel epithelium, and that the true pulp-chamber of this tooth was represented by the narrow fissure-shaped cleft between the outer and inner dentinal walls. Moral, therefore, should receive due credit for having been the first to recognize the true nature of

this anomaly, as a malformation of *one* tooth, whereas his predecessors, Baume, Busch, Salter, and Zeckendorf, were of the erroneous opinion that they were dealing with a fusion of *two* teeth.

In American literature, in 1918, we find two cases of dens in dente: one by MacDonald, one by Kirk. Both cases seem to be true representatives of the anomaly; unfortunately, however, in neither case were histologic findings reported, and we have only gross descriptions to guide us. Kirk, in describing his case—a cuspid containing within its root another smaller tooth of similar shape—stated that the crown of the inner tooth had an enamel covering of normal appearance. This statement is very unusual, because presence of normal enamel on the outside of the inner tooth has not been observed in any other case of this kind. Unfortunately, Kirk did not show sections of this tooth, and we know nothing about the tissues within the inner tooth. In addition, the crown of the outer tooth was lost. Therefore, Kirk's case was incomplete, and not as conclusive or important as the well-studied cases of Busch and Moral. The same applies to MacDonald's case. Although in all probability a true dens in dente, the description is very brief, and apparently no sections were prepared, and no facts on structural composition were indicated.

The cases of dens in dente reported by De Jonge Cohen (1918–19, 1925, and 1932) are quite different. He found this abnormality inside the root of lower bicuspids—a small root within another. By means of diagrams and photographs of specimens, De Jonge Cohen showed how this secondary inner root developed as the final stage of a process of folding-in or invagination of the mesial wall of the root along the mesiolingual sulcus (*fig. 4*). Occasionally such a secondary root may appear entirely free within the pulp space (*fig. 4*, 5). It is obvious that in such an "inner" root, formed by invagination of root wall, the cementum must be found inside of the dentin. De Jonge Cohen's cases are very similar to the "root within a root" of Miller's case. The difference between the two seems to be that whereas the inner root of De Jonge Cohen's was derived from the wall of the outer root, the inner root of Miller's was the result of an extremely deep invagination of tooth structure from the crown rootward into the root canal. De Jonge Cohen made the following statement, which explains different possibilities of formation of a dens in dente in different teeth: "The question whether we shall find the dens in dente in the crown or in the root of a tooth depends entirely on the nature of the differentiation, and thus we notice that in the bicuspids the dens in dente remains located in the lower half of the root, while in the incisors, where the dipping-in is developing from the incisal edge to the apex, naturally we shall find the described deviation in the crown also."

This opinion of De Jonge Cohen was corroborated by Erausquin, Pellegrini, and Ponte, in 1932, who described the formation of dens in dente as the result of invagination and subsequent pinching-off of a portion of the tooth, either crown or root. Their diagram, showing development of dens in dente as a folding-in of the side of a root along the interradicular sulcus, is practically identical with De Jonge Cohen's diagram.

In 1920 Lejeune and Wustrow reported anomalies of upper anterior teeth that must be considered typical cases of dens in dente. In Lejeune's case, a malformed upper central incisor of a child, the root was very short; the crown appeared (decalcified longitudinal section) to be divided into two individual crowns separated by enamel intruding from the occlusal side, and united near the apical end. As Wustrow pointed out, it was probably a case of dens in dente in an early stage of development—a dipping-in of the enamel organ into the middle of the crown, and subsequent formation of enamel in the center. Wustrow called attention to the fact that in a cross section of Lejeune's case we would find in order, from without in, the typical sequence of enamel → dentin → space (= pulp) → dentin → enamel (*fig. 1*, B). Wustrow observed an upper cuspid in a patient of 13. This tooth showed unusually pronounced development of the lingual cingulum; the lingual pit was as large as an ordinary pin-head. A radiogram revealed the presence of a tooth-shaped structure within the outer tooth. A longitudinal ground section showed that the inner structure *contained a cavity lined with enamel.* The explanation of this malformation is rather simple, if we consider all we have learned from the previous cases. The "inner" enamel is continuous with the outer enamel at and through the lingual pit (foramen coecum); this connection is not visible in Wustrow's ground section because the latter does not run through this particular plane. Therefore, the outer and inner enamels appear separated. Wustrow explained the etiology of this malformation with a diagram showing abnormal invagination of a projection of the enamel organ into the forming tooth. Thus he, too, refuted the theory of a union of two tooth elements.

A very interesting case of dens in dente was described by Gnamm in 1922. When this tooth was cut in half at right angles to its long axis, it was found that the apical fragment of the inner tooth was loose within the pulp chamber of the outer tooth, whereas the coronal fragment was firmly united with the crown of the outer tooth. This can easily be understood from study of a longitudinal section through my own case, which seems to be very similar to Gnamm's. Near the incisal end of the tooth the outer and inner walls of dentin and enamel are continuous, whereas the apical end of the inner tooth projects freely into the large pulp chamber. The enamel of the

inner tooth was found on the inside of the dentin, covering the incisal two-thirds of this inner wall. Gnamm concluded: "The histologic structure of all cases examined so far indicates that there is always only *one* pulp chamber present, around which the hard tissues are arranged in a typical, although abnormal, way. So far no case of a true dens in dente has been observed in which any trace of a second pulp chamber could be found. There is no such thing as an intermediate stage between a true dens in dente and a fusion of two tooth germs." In other words, wherever there is true fusion or concrescence of two tooth germs, we find two pulp chambers or one more-or-less-common pulp chamber, surrounded by dentin and, on the surface, by enamel; in a dens in dente, however, there is only *one* pulp chamber into which the "inner" tooth projects, the inner tooth containing enamel and dentin in inverse order; that is, enamel inside of dentin. Gnamm illustrated the various stages in the development of a dens in dente with a very clear diagram.

Herbst and Apffelstaedt, in 1928, revived the twin theory to explain dens in dente. They used the term, *odontopagus parasiticus increțus*, for this malformation and illustrated the cases of Salter, Baume, and Busch. But Herbst and Apffelstaedt apparently ignored the significance of the inverse arrangement of the hard tissues in dens in dente. For instance, in their fig. 244 one can clearly see the two fused tooth elements, each with a typical *outer* coat of enamel. Thus their classification seems to be based upon failure to differentiate between *fusion* of *two* tooth germs and *malformation* of *one* tooth germ.

Recently (1933) the problem of dens in dente was brought up by Hattyasy in his study of a large true denticle in a dog molar. Such large denticles, consisting of typical dentin and containing pulp tissue in an irregular central cavity, may at first glance appear similar to dens in dente. Up to this time I believe there has been no report of such a large true denticle in a human tooth.[4] Hattyasy's suspicion, however, that "according to the figures in Herbst-Apffelstaedt the cases of Salter and Baume are not true 'dens in dente' cases, but highly organized denticles (no enamel!)," is not justified. Had he examined the original publications instead of depending upon the reproductions in the book by Herbst and Apffelstaedt, he would have found that Salter described and illustrated the presence of enamel on

[4] In dog teeth, large true denticles do not seem to be especially rare; in one of the dogs (no. 94, for instance) examined prior to 1929 by Prof. Gottlieb and me, in our study of excessive occlusal stress, such large denticles containing living pulp-tissue were observed as an incidental finding in both upper first molars. One of the denticles was lying free in the pulp: the other showed a secondary union with the dentinal wall.

the inside of his "inner" tooth, and that "d" and "e" in Baume's illustration are marked "enamel" in the original description. The only form of true denticle that might possibly have some connection with the problem of dens in dente is the type illustrated by Fridrichovsky in his figs. 2 to 6; namely, those denticles that result from folding of the dentinal wall of the tooth. Such denticles are related, in etiology and mechanism of development, to De Jonge Cohen's dens in dente. If the folding occurs in the coronal portion of the tooth, as shown by Hattyasy in his figs. 7 and 7 a, then enamel might be developed within the fold and form typical dens in dente.

AUTHOR'S CASE

My own specimen, an upper lateral incisor of a girl of 15, had been observed and extracted by Dr. Ira P. Schofield of Chetek, Wisconsin, to whom I am greatly indebted. Before extraction the radiogram revealed a large area of bone destruction around the root-end. During the last few weeks before extraction, there had been swelling, and discharge of pus through the overlying soft tissues (p. 58). Radiographically the tooth showed a marked anomaly (*fig. 5*). Inside the large pulp-chamber there was a round radiopaque mass separated, on the mesial and labial sides, from the outer dentin by a fine cleft or fissure. Within this central mass was an irregular cavity, which apparently was completely closed and separated from the actual pulp-chamber of the incisor. The enamel covering the outside of the tooth projected upward near the tip of the crown; it then seemed to spread out and to line the incisal portion of the secondary cavity within the crown. The similarity of these radiographic findings to the illustration of Gnamm made me think of the possibility of dens in dente. I was then faced with the difficult decision as to whether to prepare ground sections or decalcified sections. The obvious advantage of being able to cut a complete series of sections made me decide in favor of the latter method, despite the unavoidable loss of the enamel. Therefore, the tooth was decalcified, embedded in celloidin, and cut into longitudinal serial sections. The extracted tooth had an abnormally thick rounded crown, making it difficult to tell which sides would normally be labial and lingual. The lingual pit, however, could be distinguished clearly. At this point there appeared to be a fine dark opening with slightly roughened edges in the otherwise normal enamel surface. The apical foramen was wide open, the canal filled with necrotic-pulp débris.

Fig. 6 shows a longitudinal mesiodistal section through the specimen. Within the large pulp-chamber, *P*, is a round body of dentin, the incisal border of which is attached to the outer dentinal wall, whereas the apical end extends freely into the pulp-chamber and, in the level of the lower third of the root, ends with a short blunted extension. In the center of this dentinal formation is a mass of enamel, bone, and necrotic soft-tissue remnants, which at the incisal end are connected with the surface of the crown through a fine opening between enamel walls. The pulp-chamber continues incisally as a narrow fissure extending between and separating the two layers of dentin. On the left side this extension can be followed well into the crown portion; on the right side, it appears to be obliterated. But examination of subsequent serial sections reveals that the obliteration is only partial, as indicated by a gap between dentin walls further down in the crown. In *fig. 7* another section of the same series runs through the apical foramen in its widest diameter, but misses the point of communication between the inner cavity and the tooth surface. The resulting picture is thus almost identical with Wustrow's illustration, with the difference that in Wustrow's case the inner cavity appears empty, whereas in my specimen it contains islands of bone in fibrous connective-tissue.

Figs. 8 and *9* are higher magnifications of *figs. 6* and *7*. In *fig. 9* the incisal end is illustrated. On both sides we see sections through a duplicature of dentin, including the incisal end of the pulp-chamber. The course of the dentinal tubules in this incisal border of dentin, while not recognizable in *fig. 9*, is indicated in the diagram (*fig. 10*). This arrangement of tubules in the dentin indicates that we are dealing with the result of a folding of one and the same layer of dentin, and not merely with a close apposition of two separate dentinal layers. The outer and inner surfaces of each dentinal wall are covered by enamel; the inner enamel is still partly preserved, whereas the outer was completely lost during the preparatory decalcification. Between the two inner layers of enamel, near the surface, is a fine strand of necrotic soft-tissue; further up in the cavity the amount of tissue is greater, and islands of bone begin to appear. The lower opening between the inner layers of enamel corresponds to the fine dark opening in the area of the lingual pit, which could be seen on the extracted tooth.

Fig. 8 shows a higher magnification of the apical end of the inner tooth-like structure in *fig. 7*. In this area the otherwise rather smooth, inner, dentin surface shows a small cusp-like projection covered with an enamel layer of uniform thickness. The most interesting thing in this area, however, is the presence of a fine central canal, and of numerous smaller peripheral canaliculi leading from the bottom of the cavity in the crown into the pulp-chamber. These communications between crown cavity and pulp-chamber explain an observation made in almost all cases of dens in dente in which a clinical history was available; namely, death and decomposition of pulp, and subsequent periapical involvement (Zeckendorf, MacDonald, Lejeune, Wustrow, author's case). The sequence of events is in all probability this: Connective tissue and bone in the crown cavity were formed before the tooth erupted. After eruption, perhaps the connective tissue remained vital for a while through the existing vascular connection with the pulp. Soon, however, bacteria invaded the tooth through the incisal opening (*fig. 9, x*), and the tissue in the crown cavity became infected and died. Subsequently the pulp also underwent infective and degenerative changes, and later periapical tissues became infected. The fact that most of the infected cases were removed in patients between 10 and 15 suggests that it usually takes from one to several years for infection to pass from the point of entry on the crown into the periodontium. This might also explain why in Moral's case—a supernumerary incisor extracted very soon after its eruption—clinical symptoms of periapical infection had not yet developed. In a case of dens in dente that develops in the way described by De Jonge Cohen (*fig. 4*), there is no possibility of pulp infection.

In *fig. 10* the findings in this specimen are reproduced diagrammatically, in longitudinal and cross section: the arrangement of the hard tissues in the cross section is identical with that in Busch's case (*fig. 1, A*).

De Jonge Cohen probably best expressed the true significance of the development of dens in dente when he said (quoted by Peckert): "It is quasi an apical extension of the lingual pit." This explanation can be verified by demonstrating the various steps of this malformation that lead from a normal tooth to a dens in dente (after Peckert):

FIG. 1, *A*

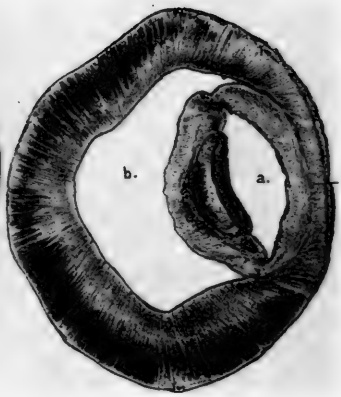

FIG. 1, *B*

FIG. 2

FIG. 3, *A*

FIG. 3, *B*

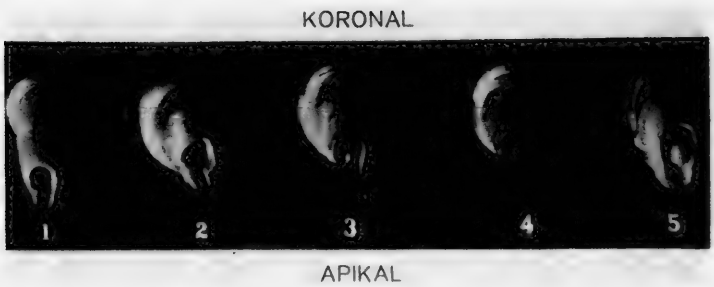

FIG. 4

Fig. 1, A and B. One of Busch's cases of dens in dente. Cross sections through different levels. *A*, Cross section through crown of twin formation. Outer tooth has coat of enamel on outside; inner tooth, on inside. Dentinal walls united in one small area. *B*, Cross section through root of twin formation, showing partial union of dentinal walls: *a*, inner tooth; *b*, outer tooth; *c*, area of fusion

Fig. 2. Zeckendorf's case of "dents télescopées." Tooth has been cut apart and shows two roots and two root-canals.

Fig. 3. Moral's case of dens in dente. *A*, Tooth cracked open longitudinally; within crown is space occupied by enamel organ. *B*, Peg-shaped inner structure taken out of large pulp-chamber.

Fig. 4. De Jonge Cohen's explanation of etiology of dens in dente. Apical ends of five lower first bicuspids showing various stages of dipping-in of mesial wall of root along mesiolingual sulcus.

FIG. 5

FIG. 6

FIG. 7

Fig. 5. Radiogram of author's case. Shows round radiopaque body with central cavity within crown of (slightly rotated) upper lateral incisor. Enamel of incisor seems to extend up into inner structure. Extensive periapical bone destruction resulting from decomposition and infection of pulp.

Figs. 6 and 7. Longitudinal sections through tooth. Section in *fig. 6* runs through area of lingual pit. *Fig. 7* shows section in slightly more labial plane, so that inner cavity appears completely separated from outside of tooth. *C*, cavity within crown. *P*, pulp chamber.

FIG. 8

FIG. 9

Fig. 8. Higher magnification of middle portion of section illustrated in *fig. 7*. Shows apical end of inner cavity and connections with pulp chamber.

D_1, outer dentin. *P*, pulp chamber containing necrotic pulp-tissue. D_2, inner dentin. *E*, enamel in inner cavity. *C*, cusp-like projection of dentin and enamel into lumen of inner cavity. *CT*, necrotic connective-tissue of periodontal origin. *B*, bone. *CC*, numerous fine canals connecting pulp chamber with cavity within crown.

Fig. 9. Higher magnification of incisal tip of section in *fig. 6*. Shows incisal end of dentinal duplicature and, in center, communication between oral cavity within crown of incisor.

D_1, outer dentin. *P*, pulp chamber. D_2, inner dentin. *D*, common dentin—dentin duplicature. *E*, enamel lining inner cavity. *X*, strand of necrotic soft-tissue leading into inner cavity. *CT*, necrotic connective-tissue. *B*, bone in inner cavity.

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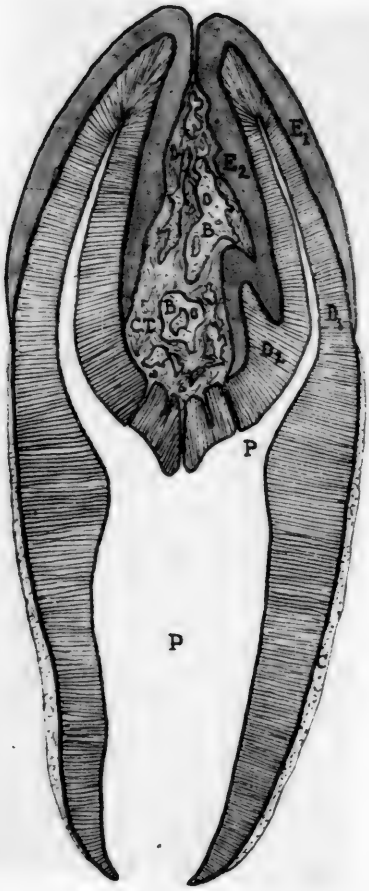

FIG. 10, *A*

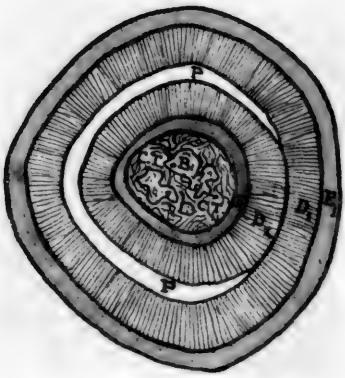

FIG. 10, *B*

FIG. 11, *A*

FIG. 11, *B*

Fig. 10, A and B. Diagrams of author's case of dens in dente

A, longitudinal section (figs. 6 and 7). *B*, cross section obtained by combining evidence from complete series of longitudinal sections. E_1, enamel on outside of tooth. E_2, enamel lining cavity within crown. D_1, outer layer of dentin. D_2, inner layer of dentin. *P*, pulp chamber. *CT*, connective tissue within crown. *B*, bone.

Fig. 11, A and B. Horizontal sections through crown of upper lateral permanent incisor, age 2. Shows development of lingual pit.

A section: through area of folding in of lingual wall of germ. *EE*, enamel epithelium. *E*, enamel. *D*, dentin. *P*, pulp. *B*, bone. *B section:* through slightly more apical level. Structures forming lingual pit seem separated from wall of germ. Arrangement of enamel epithelium, enamel, and dentin reverse of that on outside of crown.

(a) The smallest degree of this anomaly is a tooth with a markedly developed lingual cusp or cervicolingual ridge and a deep lingual pit (*Dent. Cosmos*, 1892, *34*, p. 1036, figs. 1 and 2; Endelman's Special dental pathology, 1927, p. 75, fig. 53). (b) A more advanced stage is represented by cup-shaped ("cornet-shaped") teeth, in which the lingual pit is enlarged and extends considerably into the crown; the incisal opening leading into the crown is still wide open (Zeckendorf; fig. 3). (c) In a still more advanced stage the incisal opening is almost completely closed, and invagination of the crown extends well into the root (author's case, *figs. 8* and *9*). (d) The last stage is the one in which the invaginated portion of the crown does not end with a blind sac, but is open all the way through the root, thus causing the appearance of an apex within an apex (Miller's case, or MacDonald's).

Histologic examination of the lingual pit in a human incisor shows that here occurs the process that was illustrated by De Jonge Cohen for the root of a lower first premolar (*fig. 4*). *Fig. 11* shows two serial sections in different levels through the crown of an upper lateral permanent incisor of a child of 2. In *fig. 11, A*, the lingual pit is being formed through folding-in of the lingual wall of the tooth germ. In the folded portion, the sequence is, for obvious reasons, the reverse of that of the rest of the crown, namely, enamel epithelium in the center, then enamel, and dentin on the outside. In *fig. 11, B*, a section in a more apical level is reproduced. Here the lingual pit appears almost completely pinched off from the lingual wall of the tooth; it is seen as a semicircular structure consisting—in order from within outward—of enamel epithelium, enamel, and dentin, lying in the pulp toward the lingual side of the tooth. One can easily understand how an abnormal enlargement of this lingual pit in a cross section would appear very much like *fig. 1* or *fig. 10, B*.

As far as the mechanism of formation of a dens in dente is concerned, we have to consider our present knowledge of the mechanism of tooth development. Orban has shown that the growth of tooth germs occurs on the whole in a centrifugal direction. This suggests that a dens in dente may develop not by actual *ingrowth* of enamel organ into underlying pulp, *but by relative retardation in growth of a portion of the enamel organ*, possibly at or near the lingual pit, while the surrounding dental tissues continue to expand and to grow in a

peripheral direction. The final result would then be the same as if the enamel organ were to grow into the pulp; namely, an intussusception of one portion of the tooth into another. However, it will be impossible to make a definite decision in regard to this question until suitable material (such as a dens in dente in an early stage of tooth formation) becomes available for study.

Summary of histologic findings. The case of dens in dente reported here is the result of an invagination of a portion of the crown of an upper lateral incisor into the pulp chamber, resulting in the formation of a large cavity within the crown. The outer enamel of the incisor is continuous with the enamel lining the cavity within the crown; the point of reflection of the outer and inner enamel is the lingual pit. The cavity in the crown contains connective tissue of periodontal origin, and islands of bone; at its apical end, the cavity is connected with the pulp-chamber by numerous fine canals. The pulp-chamber of the incisor, in its incisal portion, is reduced to a fine fissure, which encircles the inner body of dentin and partly separates the outer and inner dentinal layers. Beyond the incisal end of the pulp chamber, the outer and inner walls of dentin are continuous.

SUMMARY[5]

Our present general knowledge of dens in dente may be summarized as follows:

(1) The anomaly is very rare.

(2) Among the cases reported so far, in the world's dental literature, there is an almost equal distribution among upper anterior teeth—central incisor, lateral incisor, cuspid, and anterior supernumerary teeth. In addition, a special form has been described as occurring within roots of lower bicuspids.

(3) True dens in dente is characterized by presence of a tooth-like structure within the pulp chamber of another tooth. The arrangement of tissues in the inner tooth is reversed—enamel lies inside of dentin, or cementum inside of dentin.

(4) The tooth thus affected contains two cavities: the actual pulp chamber, which opens at the apex; a second, abnormal cavity within the crown, which opens near the incisal end of the tooth. The latter cavity is lined with enamel, and contains remnants of enamel organ, and sometimes connective tissue and bone of periodontal origin.

[5] See the Addendum on page 66.

(5) Dens in dente is the result of an invagination of one portion of a tooth into the other; it is not a twin formation, but a malformation of only one tooth germ.

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Addendum

All who engage in research have to depend to a large extent upon the reliability of earlier sources and references. Thus, from a study of all available literature, I believed that Baume and Salter were the first to describe dens in dente. After this manuscript had been sent to the printer, I happened to read in the work of John Tomes a description of a dens in dente of the type reported in my paper, even including presence of cementum on the enamel of the inner cavity. Tomes interpreted this malformation correctly as a deformity of *one* tooth and not as *two* teeth. His excellent description follows (John Tomes, A system of dental surgery, pp. 266 and 267; Lindsay and Blakiston, Philadelphia, 1859):

> "The defective tooth, although not necessarily the subject of any very obvious deformity, is usually a little irregular in shape, and at some part slightly enlarged. The enamel investing the crown may be, and often is, perfectly well developed; but we shall find upon the crown a slight depression, in the centre of which is a small dark spot. If the tooth be divided through its long axis, it will be seen that the dark centre of the depression is in fact the choked-up orifice of a cavity situated within the substance of the tooth, external, however, and perfectly unconnected with the pulp-cavity. If the section be a fortunate one, we shall be able to trace the enamel as it is continued from the exterior of the tooth through the orifice into the cavity, the surface of which is lined more or less perfectly with this tissue. The layer of enamel which forms the surface of the cavity is, however, thinner, and less perfectly developed than that upon the surface of the tooth, and is in some cases covered here and there with a small amount of cementum."

His illustration (fig. 98) shows a longitudinal "section of an upper tooth in which a cavity is formed external to the pulp cavity. It is lined with a thin layer of somewhat imperfectly developed enamel, and communicates with the surface of the tooth at *a*" (near the incisal edge. Ref.). It is surprising that this excellent description has apparently been ignored for a period of seventy-five years.

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Quotation from editorial comment on the desirability of withholding support from commercial dental journals.... The profession does not need the commercial journals, but the commercial journals need the profession. Backers of commercial journals are simply using the profession as a means of making money.... The more eminent the writer, the more valuable the article, the more imperative that publication be professional in every aspect and surrounding. The spirit of the past and the duty of the present is to present every valuable bit of knowledge directly to the profession, and, negatively, to refuse it to those who will exploit it for commercial gain.... There is all the difference in the world between the use of advertisements to meet deficits in the publishing of professional articles, and the contrary use of a few professional articles to justify and facilitate the dissemination of large masses of advertisements.... Dentists conscious of their professional character will only consider those journals as truly representative of their ethics which are operated on the first, and which refuse to follow the second, plan.... The professional men who are connected with... [commercial dental journals] should, in deference to their professional status, sever a connection with the commercial world which is undesirable and which tends to lower the standards of dentistry and medicine.—*Apollonian, 1934, Jan.*

Quotation from editorial comment suggesting that members of dental faculties should refrain from contributing articles to commercial dental journals. There is a clear and sharp line of demarkation between professional and commercial dental literature. The first publishes information and prints a few advertisements which help to support it—the second publishes advertisements and prints a few articles to give respectability to the advertisements.... This does not apply to the harmless trade-house journals; that is, the house organs of supply firms which keep the profession informed as to their novelties; but it applies to all those business organizations which publish periodicals filled with unreliable and uncensored advertisements of nostrums and quack-remedies. Dentists may think they receive these periodicals "free," but on second thought they will find that they "pay for it," for the advertising firms must find their advertisements lucrative, and the publishers earn their profits. The simplest way to stop this nefarious practice is for all ethical dentists to refuse to write articles for such commercial papers. The dental faculties may well take the lead and request from their members that they agree not to write for commercial dental periodicals.—*I. H.; Contact Point, 1934, Feb.*

Quotation from editorial discussion of Dr. Best's commendation of commercial dental journalism.... These magazines [commercial dental journals], according to Dr. Best, are written and edited for the profession, but it is well known they are actually published for the benefit of their supply-house owners.... From the Report of the American College of Dentists' Commission on Journalism we learn that the *Dental Survey*, of which Dr. Best is editor, is fundamentally a profit-seeking publication; and that, as one of its three owners, Dr. Best has a very practical interest in its financial success. His editorial, in which he attempts to justify and extol commercialized journalism, is therefore understandable as the effort of a person endeavoring to protect and advance his monetary interests.—*C. F. H.; Bulletin of the Hudson County [N. J.] Dental Society, 1934, Feb.*

Important Books Received

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La pratique stomatologique: Pathologie buccale, péri-buccale et d'origine buccale. By Drs. ROUSSEAU-DECELLE and RAISON. 1933: pp. 576; 6¾ × 3⅞ in.; 196 illustrations; broché 70 fr; relié toile, 85 fr. Masson and Co., Paris.

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